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INFANTILE CEREBRO-CEREBELLAR DIPLEGIA, OF FLACCID, ATONIC-ASTASIC TYPE *

L. PIERCE CLARK, M.D.
NEW YORK

INTRODUCTION

It is my purpose to place on record, for the first time so far as I am aware, a comprehensive description of a type of cerebral palsy of childhood, which I presume to name "Infantile Cerebro-Cerebellar Diplegia." Various writers have commented on the condition under the designation of cerebellar or flaccid diplegia, and very recently Förster[1] has written a paper on the condition under the head of flaccid diplegia and has given some very excellent case descriptions. Inasmuch as there has been no attempt as yet to couple this flaccid syndrome with a definite pathogeny or morbid pathologic anatomy, this paper in this latter respect may be of interest to those engaged in the study of cerebral palsy of children.

It would seem that there are two reasons for the inadequate attention which flaccid diplegias of infancy have received at the hands of clinicians. First, the spastic paretic syndrome (Little's type) has been thought to be synonymous with cerebral diplegia and cases showing any state of musculature other than spasticity, or not in accordance with this syndrome were excluded from the clinical picture of infantile cerebral palsy; second, all cases of cerebral palsy dating from birth or childhood in which flaccidity obtained have generally been considered as states of cerebral agenesis or simple defect in motor tract development. While cerebral agenesis probably often does define a condition not dissimilar to that resulting in the flaccid diplegias, it by no means is definite enough as a descriptive name to represent the series of pathological events or the nervous mechanism concerned in the formation of the clinical symptoms seen in this disease.

Just as there are varying degrees of spasticity and weakness in the voluntary muscles of the classic type of cerebral palsy of children, so there may be seen, though naturally less often, a combination of spas-

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1. Förster: Deutsch. Arch. f. klin. Med., 1909-10, xcviii, 216.

ticity and flaccidity in one and the same case. Often side by side in the one limb or segment of a limb flaccid muscles may be found in some otherwise spastic cases, and occasionally one of the lower extremities may be found flaccid and the other spastic, or still more rarely, the upper extremities may be flaccid and the lower spastic. The not infrequent freedom from involvement of the upper extremities while the legs are spastic in a common diplegic is not lost sight of in making this statement. As the clinical material is more carefully analyzed and proved by autopsy study we shall learn to distinguish both the extent and depth of lesion in the palsied brain and possibly arrive at a better prognosis than now obtains.

The flaccid (atonic-astasic) type is even now thought to be an extreme rarity by many. One finds, however, many incomplete descriptions or mention of the condition throughout the extensive literature of cerebral diplegia without the state being diagnosticated as such, and so far as I am aware no one has attempted to explain the lesion underlying the condition or its pathogeny. Förster has called particular attention to its failure of diagnosis and the difficulty of explaining the character and type of the lesion which causes the flaccid syndrome. In 1899, Batten[2] cited the fact that pediatricians not infrequently encountered cases of ataxy, incoordination, disarthria and hypotonia in children during earliest infancy. The cases which he recorded differed considerably from each other, but he thought he saw a sufficiently marked similarity in all the cases of his group-study to give it the name of cerebellar diplegia. In his type, however, it will be remembered the condition was for the most part transitory, or at least largely removed in the course of a few years. His last case of record, however, remained nearly stationary and without material improvement, and was also attended and followed by nearly complete mutism and imbecility. Its description nearly paralleled those which are the subject of this paper. Inasmuch as Batten's cases never came to autopsy, he had to content himself with postulating the nature and seat of lesion. He held the essential lesion to be a vascular injury to the cerebellum, its peduncles, and sometimes to the mid-brain itself. At that time our present knowledge of more exact functions of the cerebrum and cerebellum, especially their corelationship in disorders of equilibrium and regulation of voluntary movements, had not been attained, and his explanations were not so satisfactory as they might be. The whole subject does not seem to have been pursued further by him or others.

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2. Batten: Trans. Clin. Soc., London, 1902, xxxv, 205.

TYPE OF CASES EMBRACED IN INFANTILE CEREBRO-CEREBELLAR DIPLEGIA

The type of case embraced in the flaccid syndrome is very striking So striking, in fact, that had it not been entirely excluded from the nosological group of palsies of children and classed with agenetic defects, it must not have lacked a more definite study long ago.

The developmental history of the affection is as follows: The child is usually born without asphyxia in a non-instrumental delivery. The intrauterine life of the child in the majority of cases seems to have been uneventful. Only in rare instances has there been good evidence of trauma to the pregnant mother during gestation of the after-affected child. One of my cases was an instance in point. Even under such circumstances many injuries of the like or even more serious character have been of frequent report without having entailed any wide extent of neuraxal lesion as seems to be in evidence in our palsy type here described. The mooted points in its pathogeny will be considered later, in connection with and following the probable pathology.

Usually nothing abnormal in physical and mental make-up is noticed in the child for the first few months after birth. The unusual becomes evident only at that age when the child attempts movements of its own volition, at about 1 year or 18 months of age. It is then noticed for the first time that the child cannot sit up or hold up its head, much less stand. The motility of the arms and legs is normal. The child commonly lies on its back with lower extremities bent considerably at the knees and hips and strongly abducted and rotated outwardly. The legs can be easily extended, and once done, remain so without position. It is then seen that the whole musculature of the body is relaxed. There is no atrophy present, nor does any succeed at a later date. The electrical excitability remains normal. The most striking symptom is the enormously exaggerated mobility of the joints. Up to this point in the description the condition is not so very dissimilar in appearance to a myotonia congenita to which infantile cerebro-cerebellar palsy may be allied in some of its essential features in defect of muscle tonus. However, the analogy ceases with the similiarity in loss of muscular tone. Further examination of infantile cerebro-cerebellar diplegics shows that there is no muscular resistance and the different segments of the limbs can be maintained easily in very arbitrary positions without pain. (See photographs.) The positions in which the limbs of the child can be placed recall those actively assumed by contortionists. This extreme hypotonia has naturally an intimate relationship with the absence of voluntary motion and station. In later years, as the child grows, an ataxy of cerebellar type is superadded and incoordination of all the extremities is marked whenever the child attempts to stand or walk.

Physical examination reveals a wide range of alterations of the reflexes. From increased knee-jerk to a marked diminution or rarely an absence of the same may be noted. As would be expected, ankle clonus is absent while the Babinski reflex may or may not be present. Usually the mental deficit amounts to idiocy or a low degree of imbecility, at least. The mental state undergoes little change with advancing years. There is always a marked speech defect, in the majority of instances amounting to mutism; in others, two or three words only being spoken. While an intrauterine pathology seems likely for this palsy type, not infrequently one finds cases of extrauterine insult either at the time of birth or as the result of a meningo-encephalitis of wide extent. In such cases, though the child may have been able to speak fairly well, a more or less complete loss of speech and idiocy succeeds. As would be expected, the later after birth the brain insult is sustained, the less typical is the clinical picture as above outlined. Still further, one sees other transition cases of flaccid-spastic type which rather insensibly shade out into the classic spastic syndrome, pure and simple. In the transition cases, as not a few of Förster's and one or two of mine herein illustrated show, in addition to spasticity, there are choreic movements somewhat resembling the coarse intention tremor of disseminated disease superadded; even athetosis may be encountered in the impure types. One of Förster's cases had an opisthotonos of the neck and back, whereas complete flaccidity obtained elsewhere in the body. In one of his atypical cases of flaccidity he found a Babinski, distinct spasticity in the lower extremities and increased reflexes. Most of the spasticity in these flaccid cases is developed in looking for resistance.

However, no one should confuse these atypical cases with the classic type of infantile cerebro-cerebellar palsy, the object of our study here. The above digression is made merely to show that under varying conditions of causation very nearly identical nervous structures may be involved in two disorders of the neuraxis essentially differing in pathogeny and anatomical pathology.

The prominent and constant symptoms in all cases, as previously mentioned, are the pronounced flaccidity, the mutism and idiocy. These symptoms establish the disease type. All the other symptoms may vary in special cases, but the symptoms just enumerated are constant. Later, we shall try to make plain the reason for the same.

The positions of the arms and legs are not fixed by proper muscle tone. There is discontinuity of movements, shown in the absence of a cadence of the rhythm and release between the flexors and extensors. A dysmetria, or a lack of proportionate adjustment of the movements to the object in hand, also occurs. If the limbs are left to themselves they follow the action of gravity, and when let fall from a lifted position

behave in as lifeless a manner as a lifeless extremity of a recent hemiplegic. Yet even here the analogy fails of further comparison, for the passive motility of the limbs in infantile cerebro-cerebellar diplegia is far greater than in any hemiplegic, and the extremity offers no resistance to extreme flexion or extension. The knee can be hyperextended and then the heel may be easily carried back to the buttocks or placed on the shoulder of the same side with no expression of discomfort whatever. (See photograph.) The increased passive mobility is equally observable in all joints and in all their movements. Even though one bear in mind the frequent looseness of joints in children, one notes here a distinct pathologic flaccidity in muscles and joints.

As a natural corollary to the flaccid joints and muscles one understands why these children cannot stand. Many of the children can assume no posture at all and always collapse like a jointed doll toy. In some respects the foregoing phenomenon is not unlike the temporary flaccidity not infrequently seen in spastic diplegics after extensive dorsal root section has been undertaken. In the severest grade of the flaccid diplegics the child cannot support the head on the trunk nor hold the trunk erect in the sitting posture. In some mild cases, as the child grows older, one may hold the child by the armpits (see photograph), just permitting the feet to touch the floor. This procedure appears to induce a sort of epileptoid trepidation in the lower extremities, an incoordinative dance. The movements are wild and bizarre like a jumping-jack toy. The effort, be it attempted a volitional standing, or simply a reflex, quickly exhausts the child, fatigue being rapid, when the movements become labored and feeble. After a short period of rest the movements may be reinvoked as before. Rarely in any of these children as they grow older is there an entire absence of voluntary motion of some sort, and in most of them considerable motion is seen in earliest life. Such movements are seen better when the child sits, being held, or better still, when it lies in bed on its back. This statement, however, should not militate against the former statement that in the first few weeks of life these children are, at least in the vast majority, passive and inactive in all movements. This passive state is frequently overlooked as a similar inactivity is not unusually seen in obstetric and pediatric experience where prolonged or difficult labor has resulted in the birth of asphyxiated or puny children. The early observations of the child's inactivity are brought out in sharp contrast by the suddenness with which it begins in the course of a few weeks to show marked and unusual ability to move the limbs strongly. This fact is frequently commented on by the mother of the child, who is much mystified at the further lack of the child's motor development. It is also in sharp contrast to the gradual acquire-

ment of power in spastic diplegic cases. The point is especially called attention to here to show the remarkable effect on even slight development of motility on the part of the growing cerebellum when the cerebellar "brake" is absent or withheld. One must discriminate between purposive movement and those automatically made, as here even in severe cases, while the child is lying recumbent, the head may be moved backward and forward, and exceptionally the head may be jerked about violently, but fixed positions implying purposeful and therefore coordinative movements are never possible. Some of the muscles, on command, may be felt to grow rigid with voluntary effort, but the latter are unproductive in locomotive effect for the other muscles necessary for a voluntary effort refuse to act. This is essentially a disorder of the cerebellar function.

The entire picture of flaccid diplegia or infantile cerebro-cerebellar disease may be summed up as follows:

The defect in active or voluntary motion is due to a complete lack of cooperation on the part of the antagonists and their collateral synergistic muscles, while the immediate flexor or extensor muscles may act with energy. This, too, is an essential defect in cerebellar function.

In looking for a comparison for the flaccidity in this disease, one is strongly reminded of that seen in poliomyelitis, or, better, tabes, though more pronounced in infantile cerebro-cerebellar diplegia. Probably the underlying cause for flaccidity in the two, tabes and our subject, is not so very dissimilar, as we shall see in the theoretic portion of this paper.

The disorder of motility, the wavering tremor, is much like a minor chorea. That the excursion of movement is large is due to two factors, the hypotonia and the dysmetria and unequal control in opposing muscular forces. The condition, as might be expressed, is seen most exquisitely in those receiving their disorder in earliest intrauterine or intrapartum life. The injury and wide destruction of the lesion in cerebral motor function accounts for the persistent disorder, as the anterior cerebrum, the frontal, parietal and temporal lobes can substitute cerebellar functions, and many functions of the cerebrum, as regards motility, reciprocate with cerebellar ones. We must permit in the disorder under study as wide a latitude in clinical expression as that of the ordinary type of Little's syndrome or cerebral diplegia; for in both we are dealing with the negative and positive manifestations of neuraxal symptoms — a diseased nervous system trying to compensate and right itself while in process of development — and especially, too, in that part of nervous functioning, that of motility, acquired in greater part after birth. These are essentially cerebellar functions *par excellence.*

PATHOLOGY AND PATHOGENESIS

What is the connection between these symptoms and an intracranial lesion? Actual proof and study of pathological material we have none. Förster's two autopsied cases are beside the point as they were not classic flaccid cases. The mental defect, idiocy, did not obtain, nor was there complete mutism. They had epileptic convulsions. In passing, I may say that one of mine had convulsions in the first two years of life, but fits have now been absent for two years. One still has fits, and another has trembling fits, a form of generalized cloni as has been noted in recorded cases of cerebellar agenesis. When one searches for similar clinical phenomena in other nervous disease states, one is at once impressed with symptoms recorded in cases of cerebellar disease, particularly the atonic astasic form of cerebellar ataxy, but the history and evolution of the two affections radically differ. For instance, the latter in its pure form shows no extreme extensibility of the muscles and looseness of the joints. There can be no doubt, however, that somewhat similar structures, in part at least, are involved in the two affections.

In closing the clinical portion of this paper on infantile cerebro-cerebellar diplegia, I submit we have to deal here primarily with an intrauterine disorder, which may either be the result of inflammatory or hemorrhagic injury, or, which is probably more plausible, an agenetic disorder of the cerebellum and a wide and extensive defect of the forebrain, especially the frontal, parietal and possibly the temporal lobes of the cerebrum, as a result of which hypotonia, dysmetria, ataxy, incoordination, mutism and idiocy result.

Let us review briefly the more recent experimental and clinical evidence of cerebro-cerebellar symptoms to make clearer the pathologic anatomy of our disorder of the infantile cerebro-cerebellar diplegic.

THEORETIC CONSIDERATIONS OF THE RELATIONSHIP BETWEEN CEREBELLAR FUNCTIONS AND THE FOREBRAIN, AND THE BEARING WHICH THESE HAVE ON THE CEREBRO-CEREBELLAR PALSY OF CHILDREN

While it is generally conceded that the cerebellum plays little or no part in the organic functions of the body, of sensibility or intelligence, there are few functions in life concerning the relation of movements in which the cerebellum does not enter. As regards the mildest grade of intelligence defects occasionally noted in cerebellar disease, though the case be a specific and limited lesion of the cerebellum solely, the intellectual defect would be understandable by finding a certain mental deficit whenever there is a lack of mechanical adaptation of bodily movements from which the psyche receives no small impetus to its proper development; the individual subject might be so engrossed in minute

and automatic adaptations of bodily movements in cerebellar disease as to narrow the limits of the actual field of experience from which the psyche gains its greatest incentive for growth.

In considering the relationship of the cerebellum to motility, it must be said that some investigators still believe that this organ is the center of energy, or reinforcement of muscle tonus, though the majority of writers seem to entertain the idea that it is purely a center of coordination of all bodily movements. Still others limit its rôle to that of equilibration only. It is fairly well known that the peripheral excitations are gathered together in the cerebellum on their way to the cerebral cortex. It is therefore conceivable that the cerebellum may be an active organ in elaborating or controlling muscle sense; at least to that point of making it a relay station for these incoming excitations and thus it would necessarily play a rôle in the variations of muscle tension so essential for proper equilibration.

As a matter of anatomic moment, we know that the gray substance of the cerebellum and pons has a crossed function, while the nuclei of the latter communicate directly with the cerebral cortex, especially the forebrain of the same side. This association is made principally by the frontal bridge tract through the crus cerebri to the frontal, the ascending parietal lobes and the paracentral lobule. A portion of this tract known as "Türck's bundle" takes its specific origin from the middle segment of the second and third temporal convolutions exclusively. Therefore we see that there is a close functional relationship between the cerebellar and the cerebral cortex. The cerebral zones here considered, which are projected on the cerebellar cortex, are primarily sensori-motor zones. The second and third convolutions, however, are ordinarily considered as centers of impressions of labyrinthian origin which pass from the middle cerebellar peduncles. The interrelationship of certain portions of the cerebrum and cerebellum is proven in part at least by comparative embryological studies which always show a parallel between the development of the pons, the median cerebellar peduncle and the cerebral cortex.

Even though both the hemispheres of the cerebellum are destroyed in animals, it is known that reeducation takes place in time by means of the reciprocating functional powers of the cerebrum. In cases of cerebellar agenesis, coinciding with a cerebral defect of the forebrain, mutism is almost invariably found in both experimental and clinical studies; dogs do not bark and children do not speak after lesions of these parts. The reflexes in total destruction of the cerebellum are increased, the "brake" on the reflex arc is removed. However, whenever a wide destructive lesion of the cerebrum is entertained, especially in the forebrain, together with cerebellar ablation, the knee jerks are markedly diminished or lost. This variation in reflexes under experimental conditions explains

much of the wide variation seen in the reflexes of the clinical types which we have under study. Moreover, when both middle cerebellar peduncles are excised in animals, they lose power to stand or walk.

In applying experimental evidence on the cerebellar functions to explain our clinical type, we must proceed with due caution, as the symptoms of hemorrhage and softening in a fully evolved neuraxis are quite different from those of lesions of a slow and progressive disorder, and, moreover, both groups of symptoms are still more variant when they occur at birth, in the cerebellar organ, which is still outside the realm of every-day use in the new-born child.

Many of the cerebellar experiments cited in literature are incomplete, and in others the lesions are not completely confined to the limits of the

Fig. 1.—T. S., 5 years old. At 1 year of age had fever and convulsions; five weeks sick; lost all speech and slight ability to stand. Now all extremities are very flaccid and hypotonic, greatest in legs, right arm and neck. Bilateral Babinski, knee-jerks +. No ankle clonus. Coarse incoordinate movements in arms, legs, head and trunk.

cerebellum alone. As is known, the symptoms following cerebellar disease are staggering gait, widening of the base of support and a disturbance of equilibration. The patient falls to the side or backward, according to whether one cerebellar hemisphere or both are equally involved. To this picture may be superadded an intention tremor or cloni in the upper extremities and certain incoordinative and ataxic movements in the lower limbs and rapid fatiguability. Vertigo and various ocular disturbances as well as nystagmus are less common. While most authorities agree that a real paralysis, such as that of ordinary hemiplegia in the adult, cannot be produced as a result of disease of the

cerebellum or experimental ablation of the same, most writers are agreed that a certain type of motor defect which *amounts* to paralysis may be seen. The defect, too, is seen in *all* muscles in contra-distinction to that seen in ordinary hemiplegia, where special groups of muscles, such as a paralysis of dorsal flexors, flexors of leg, extensors of hand and external rotators of the arm are chiefly if not exclusively affected. Further, in the motor disability of cerebellar disease there is always an absence of

Fig. 2.—T. S. Showing flaccid ankle-joints.

contractures. Finally, the reflexes are exaggerated *without the occurrence of spasticity,* and in the pure uncomplicated type of cerebellar injury or disease there occurs no epileptic trepidation nor Babinski. The foregoing facts all point to a better understanding of the anatomico-pathological condition which has been postulated for our type of infantile cerebro-cerebellar diplegia.

In reviewing the clinical reports on cerebellar disease, one is impressed with the entire absence of symptoms in some cases even though a complete cerebellar agenesis has occurred. Cambetti's case is an instance in point; the lesion was clean cut, showing an entire absence of the cerebellum without any other defect. The child developed slowly, was backward in all essential organic movements and spoke with diffi-

Fig. 3.—T. S. Wide base of support. Cannot stand alone or walk.

culty and hesitation. In Anton's case, that of a little girl of 6, all her movements were slow and incomplete. She sat up, stood and walked only after 4 years of age. Even then she was unable to walk freely; she moved about only by artificial support. Incoordination, staggering and frequent falls persisted. Rapid fatiguability was very marked. The speech was not very clear, but no tremor was in evidence, and the knee-jerks were exaggerated. In one of Shuttleworth's cases there was gen-

eral muscular weakness, tremor of the hands and arms, and the child remained an imbecile. Borell's case showed an extraordinary bending of the legs in walking (due to looseness of joints). In all cases there have been motor disturbances and considerable hypotonia. In all, there has been an absence of the rhythm or cadence of normal movements. The peculiar defect in attempts at walking in cerebellar patients is quite characteristic. The foot is frequently advanced ahead of the trunk, while the latter does not follow up the attempt at progression by the leg, consequently there results, as in my cases, a sort of paddling movement of the feet without trunk advancement which is necessary for normal bodily progression.

From a careful search in the literature, one is impressed with the fact that *bona fide* cases of primitive atrophy of the cerebellum, which

Fig. 4.—T. S. Showing hypotonia of knee-joints.

would be most pertinent to our study, are very rare, and if one includes those cases in which other parts of the neuraxis are absolutely healthy, the case material is exceptional. There can be little doubt that a *lack of proportion in volitional movements*, seen in nearly all cases of infantile cerebro-cerebellar diplegia, a phenomenon named dysmetria by Thomas and Babinski, is a sure sign that the cerebellar portion of the neuraxis has been seriously crippled in this palsy. If one examines these children carefully he finds the essential defect in volitional movements is due to a contraction of the muscles in several stages and not in a tonic fashion as normal muscles are; therefore, a sort of cloni occurs which may amount at times almost to an epileptic crisis of cloni, as noted in one of my group. This speaks for a serious damage to the coordinating func-

tion of muscle rhythm which is presided over by the cerebellum. The rapid fatiguability well shown in my material is to be accounted for, not on the basis of a weakness or a paralysis, but therein the automatic effort of repeated testing out on the child's part of large groups of muscles calls for a great expenditure of energy and rapid fatigue results.

It has been found that substitution of the function of one lobe of the cerebellum by the other occurs up to a certain point, while in the absence of both hemispheres, as previously mentioned, the frontal lobes contribute an important help in maintaining equilibrium. An interesting case which was proofed by autopsy and found to be olivo-fronto-cerebellar

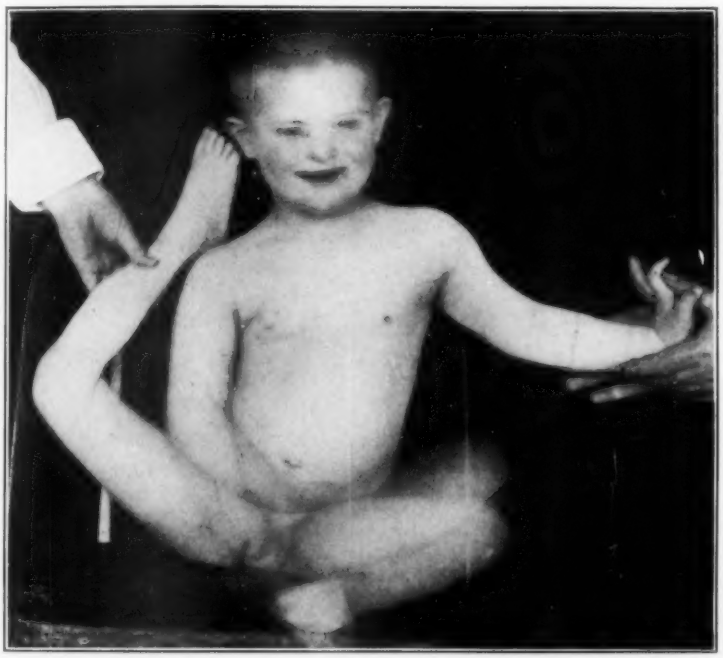

Fig. 5.—F. N., 8 years old; no history of palsy or injury. Never talked nor walked. Classic flaccid hypotonia of muscles and joints. Some ability to stand supported, but very marked incoordination; most pronounced in right leg. Wildly paddles legs when walking is insisted on. Apparently understands two or three simple commands. Idiocy. No Babinski, and knee-jerks —. No ankle clonus.

atrophy, showed disorder of gait and station, nystagmus and scanning speech; but there was here also a bilateral atrophy of the cerebellar peduncles, while the pyramids remained absolutely intact. The patient was therefore not really paralyzed, and there was no real motor weakness. All this goes to prove that any disorders of movement in atrophy of the cerebellum are accounted for by interruption of the fronto-bridge tract, and, secondly, if the latter remains intact the cerebral cortex may sub-

stitute in a large measure the defective functioning of the cerebellum in regulating muscle movements. The substitution is more easily made, and the more complete, the earlier it takes place in the development of the neuraxis, before the nerve centers have been affected by subsequent perverse habits. The individual differences in range and degree of the lesion explain the difference in the degree of recovery in our cases. It is a common fact that whenever there is very wide area of the motor zone diseased dating from infancy, the opposite cerebellar lobe is atrophic as well as the pons of the opposite side, and the olive of the same side as that of the cerebral lesion. This is very well shown in a case of my own reported some years ago in an epileptic girl. In such case the atrophy of the cerebellar cortex, the middle cerebellar peduncle and degeneration

Fig. 6.—F. N. Shows hypotonia of legs of same child, who is easily placed in an attitude which would be very uncomfortable to a normal one.

of the peduncular path followed into the gray substance of the pons, the pontine nuclei. As a system, this fasciculus which had degenerated is known as the cerebro- or fronto-cerebello-pontine tract. The cerebral cortex of the frontal, parietal and temporal regions is projected on the lateral lobes of the cerebellum of the opposite side and also to a slight degree on the lateral lobe of the same side by the intermediation of the cerebral crus and the pontine nuclei. A consideration of the spinal medullary centers which are projected on the vermis proves that to some degree the functions of the vermis and the lateral lobes are different. It is the first system which suffers a defect in the infantile cerebro-cerebellar diplegias. This system, once disordered, as in infantile cerebro-cerebellar diplegics, would bring about a defect in equilibration primarily.

Finally, we may summarize our subject as follows: We have here under consideration an extreme defect disease of the cerebro-cerebellar function with relationship to bodily movements, of an unknown pathogenesis (probably based largely if not solely on a developmental defect) which is for the most part intrauterine in origin and which we find expressed in clinical manifestations of atonia, astasia, hypotonia, mutism and idiocy. We believe it constitutes a distinct type of palsy of infancy. The disorder not only affects isolated movements, but particularly the *association* of movements or the motor synerges so-called. The movements by themselves as shown in the type are not incoordinated as in tabetics, but are characterized by dysmetria and discontinuity. When-

Fig. 7.—M. D. Showing hypotonia of wrist.

ever the child's movements relate to the maintenance of an attitude, especially in sitting or standing, there is instability, wavering from side to side, or astasia. The children show great disturbance of the reactions to equilibration, due to the fact that the defective cerebellum fails to effect and hasten the reestablishment of equilibrium, much the same way as it fails to make isolated movements precise and regular. Again, the defective cerebellum fails to apportion the measure and the continuity of movement. There is instability and imperfect reactions to equilibration by a special tonic action. This latter function is co-governed by the cerebrum and to some degree by peripheral stimulation. The defect in cerebellar function shown in hypotonia is not compensated for by a dis-

eased cerebrum. The latter organ being unable to substitute or take up the cerebellar function there results a persistent dominance of hypotonus throughout the life-picture of this type of palsy. Again, as exquisitely shown in our clinical type, the suppression of the cerebellar functions in the condition or disease under study does not produce a palsy *per se* as ordinarily understood. Motility of the limb remains. It is never

Fig. 8.—M. D. Hypotonia shown in child sitting in extreme "tailor fashion."

abolished entirely, as some movements or attempts at equilibration probably remain because some degree of substitutional power of the cerebrum is still possible; the greater part of its loss, however, is largely in evidence in this unique disease.

SUMMARY

We may here briefly summarize the points brought out in the foregoing paper. (1) Infantile cerebro-cerebellar diplegia of the flaccid, atonic-astasic type is a relatively rare disorder, which has largely an

unknown intrauterine causation, but which results in a permanent disablement of the motor functions of the cerebellum and forebrain, resulting in (2) a state of mutism and idiocy accompanied by atonia, astasia, dysmetria, incoordination and a generalized palsy in effect.

ADDENDUM

While the data on cerebellar disease is already extensive, that which has a more or less distinct bearing on our type of associated palsy of brain and cerebellum is not so great but that we may be permitted to give a few abstracted case-histories which illustrate some of the special points. It is hoped that citations of this kind may signally assist the

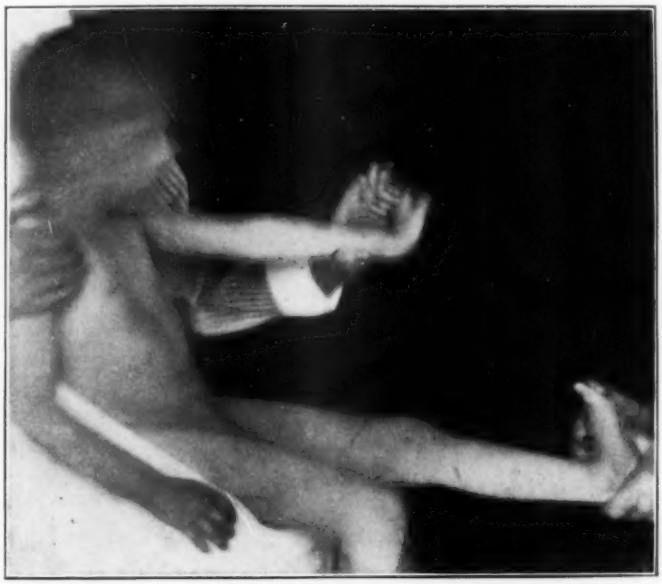

Fig. 9.—M. D., 4 years old; youngest in family of five. All others healthy. Patient born normally at term. Normal mental and motor development (?) until 5 months' old, when, after a few days of fever, diarrhea and convulsions all extremities became limp. Fits continued for two years, severe and frequent. When mother was three months pregnant with patient she had a fire fright and fell downstairs. Child has never spoken, and is a profound idiot. Never walks but often tries unsuccessfully to stand. Cries continually. Bilateral Babinski, vasomotor stasis in hands and feet marked. Knee-jerks + and no clonus. Lower extremities flaccid, hypotonic in muscles and joints. Marked incoordination.

inquirer to orient himself, as it were, regarding the relative clinical and pathological position which the new type of infantile cerebro-cerebellar palsy should occupy in the brain palsies of infancy.

In looking over the monumental work of Freud, one is impressed that at the time his special work on infantile cerebral palsies was written,

some twenty years ago, the ordinary conception of a flaccid palsy was indicative of an abortive case, and in such afflicted individuals one might expect them to recover (like Batten's cerebellar diplegia of 1898-9). Freud regarded the existence of a permanent flaccid palsy as improbable, as a spastic or rigid state *might* have existed at some period unobserved.

Fig. 10.—M. D. Shows typical straddling attitude of child unable to walk or stand alone. Note blur in photograph from incoordinate movements of head and left hand. Back and head supported.

In Adler's 134 autopsies in cerebellar lesions no cases occurred in the newly-born, or even in the first two years of life, and very few in children of any age.

That cerebellar agenesis or atrophy *per se* cannot cause a flaccid state is shown by the fact that some individuals born with a rudimentary cerebellum seem to show no motor disorders. On the contrary, if the cerebellar atrophy is secondary to a cerebral lesion, or if the cerebellum

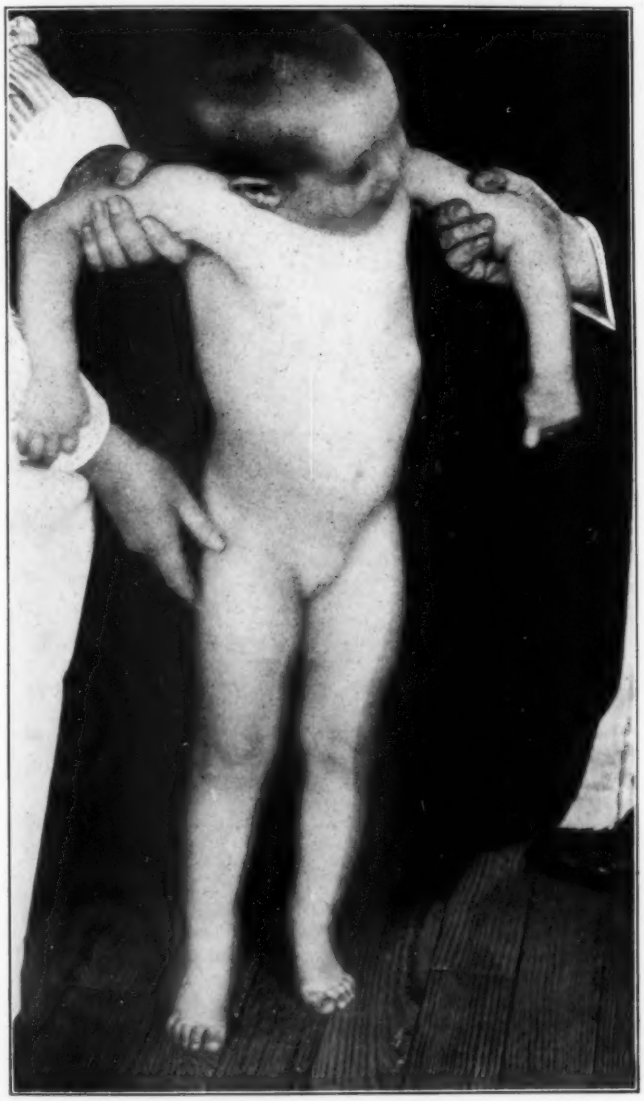

Fig. 11.—L. S., 4 years old; idiot; cannot talk, walk nor stand. Flaccid and hypotonic in all extremities. Marked incoordination. "Dancing doll" type when held standing. Note inability to hold head erect. Knee-jerks +. Bilateral Babinski. No ankle clonus.

and cerebrum are both agenetic, very complex symptoms present themselves. A typical illustration of each will be given.

The first case is one of congenital absence of the right hemisphere, reported by A. Pearce Gould.[3] The man lived to be over 80 years of age, and had worked for sixty years for one family as a gardener. Nothing

Fig. 12.—L. S. Shows result of an attempt to get the child to walk. Head is in incoordinate associations. Legs held wide apart, the right one bearing little weight is in dancing vibratory motion.

else was known of him, as shortly before death he had been moved to an asylum where he was unknown. The left half of the cerebellum was

3. Gould, A. Pearce: Trans. Pathol. Soc., London, 1881, xxxiii, 6.

normal, and there were no evidences of disease in the vicinity. That the condition represented an agenesis and was not the result of some ancient lesion well appeared from the aberrant course of the lateral sinus, which occupied much of the cerebellar fossa; the flattening of the bone externally, the negative evidence as to tumors, abscesses, etc., and finally the normal quality of a rudiment of the left hemisphere. This agenesis had led to no serious motor disorder.

In explanation of the lack of motor defect, it may be fairly urged that this case but illustrates the point made in the theoretic portion of this article; one sound lobe of the cerebellum may compensate for even an entire absence of the other lobe. This case with retention of normal motility is quite similar in all essential respects to that reported by Hitzig.[4] The patient, female, was first seen at the age of 32 years. The mother stated that she had never had any disturbance of motility, had learned to walk at the proper age, and could leap and dance. She was backward in school studies, but married in later life. Nine months before consultation she developed paralytic dementia from which she died. The author shows elaborately that the case was one of agenesis, not of secondary atrophy. He believed that the cerebrum probably acted vicariously in these cases. The two cited cases bear out Luciani's[5] contention that agenesis of one-half of the cerebellum causes no symptoms, while in agenesis of the whole organ incoordination and hypotonia surely follow.

In one-half the cases recorded in literature one finds bilateral incomplete agenesis in a latent form and in the other half ataxia, astasia in lower limbs, and frequently in the arms and hands also. Luciani expressly states that the latent agenesis or defects dating from embryonal life the cerebrum compensates for the cerebellar defect, although he throws no new clinical light on our type of palsy in the newly-born. A very remarkable case showing how the other portions of the neuraxis may show compensatory hyperplasia in bilateral defect of the cerebellum is reported in clinical and autopsy detail by Anton.[6] As it is one of the most exhaustively reported cases on record of its sort, I will give it in brief abstract.

Mother of child delicate; father tuberculous; gestation normal. Labor easy (infant very small) and finished in an hour. Infant nursed normally and later was fed with a spoon. It was noted very early that swallowing was awkward, causing coughing spells. The movements of the limbs and trunks were learned slowly and imperfectly. The child was in her fourth year when she learned to sit and stand, and she clung

4. Hitzig: Arch. f. Psychiat., 1884, xv, 266.
5. Luciani: Ergeb. d. Physiol., 1904, iii, 1, 259.
6. Anton: Wein. klin. Wchnschr., 1903, xvi, 1349.

to objects for support. She could never walk backwards, and all movements were weak. She became quickly exhausted after efforts of any sort. Speech was defective, low and infrequent. She was somewhat apathetic and greatly irritated by certain sounds. There was no tremor of the hands, but they became exhausted so quickly that objects were passed from one hand to the other and back again. Attention was difficult to fix. At the age of 4½ years she died of acute laryngitis. A full autopsy report is given. The absence of the cerebellum was thought to be due to porencephalic softening.

The parts which showed the compensatory over-development were the pyramidal tracts, the nuclei of the posterior columns, the gelatinous substance of the trigeminus with its sensory nucleus and locus ceruleus. The thalamus and corpus striatum were well developed and showed no sign of degeneration. The cerebral cortex was thicker than normal — a relative hyperplasia.

Ewald, Thomas and others have proved that compensation of this sort does occur. It is, of course, only partial, but may improve progressively.

From the above brief citations from the literature of our subject one is impressed that the whole subject is entirely covered experimentally and pathologically, and it proves that (1) unilateral agenesis is compensated for by the remaining intact lobe, and (2) that bilateral agenesis may be compensated for by the cerebrum. Evidence is not yet forthcoming to show in cases (like ours) in which agenetic defect of both cerebellum and forebrain is postulated to exist, in what way the multiple lesion is induced, whether the cerebellum or the cerebrum suffers the initial damage or whether both are simultaneously injured, and how. The pathogenesis of the disorder still rests in the darkness and mystery of intrauterine pathology.

In reviewing the literature of cerebellar disease, I wish to give proper credit to the excellent work and résumé of Dr. André-Thomas[7] of Paris. I have consulted the original articles on cerebellar research and have found his exposition so complete and satisfactory for the purposes of this article that I have borrowed the substance of many of his conclusions of the co-functioning of brain and cerebellum where such have been particularly pertinent to prove the hypothesis of my thesis. All the statements in this paper based on the morbid anatomy and physiology of different workers on cerebellar disease have received the critical sanction of Thomas, as above noted.[8]

84 East Fifty-Sixth Street.

7. André-Thomas: "Cerebellar Functions," Nerv. and Ment. Dis. Monograph No. 12. Jour. Nerv. and Ment. Dis., New York, 1912.

8. The clinical material shown in the photographs illustrating this article is drawn from my service in the Randall's Island institutions for paralytics and mental defectives.

PAPULO-NECROTIC TUBERCULIDES IN INFANTS

BENNO M. WRONKER, A.B., M.D.
NEW YORK

"Papulo-necrotic and papulo-squamous tuberculides (Boeck) are small, round, slightly raised, rather hard, flat papules ranging in size from a pinhead to a millet seed, of red to reddish-brown to livid brown color, often presenting on their surface a small dry crust which is easily removed, leaving a whitish, central depression with occasionally a small bleeding point in its center. If put on the stretch they have a glistening appearance." (From Hamburger's[1] description). The lesions generally appear in crops and heal spontaneously in from four to eight weeks.

HISTORICAL

Before going into a general discussion it will probably be of interest briefly to review the history of the relation of these and other tuberculous lesions to tuberculosis. Before Koch's discovery of the tubercle bacillus only the ulcerative lesions of certain types were classified as tuberculosis of the skin, although it was noted that individuals with tuberculosis were prone to certain forms of skin diseases. After this period the nature of that large category of skin lesions now classed under the head of tuberculosis of the skin began to become known and the following requirements were determined on as sufficient to place a lesion in this category:[2]

1. The finding of bacilli (a) in sections, (b) by culture, (c) by inoculation tests.

2. Tuberculin reactions.

3. Histological findings.

4. Occurrence of other manifestations of tuberculosis.

Of these the inoculation tests and the histological findings are of most importance.

There were three routes of infection considered at that time.

1. From without by (a) autoinoculation, or (b) from external sources.

2. Through the skin from subcutaneous lesions.

3. By metastases, through the blood- or lymph-stream.

The following classification was then established of lesions known to be of bacillary origin: 1. Lupus vulgaris. 2. Scrofuloderma. 3. Tuberculosis cutis verrucosa. 4. Tuberculosis miliaris ulcerosa.

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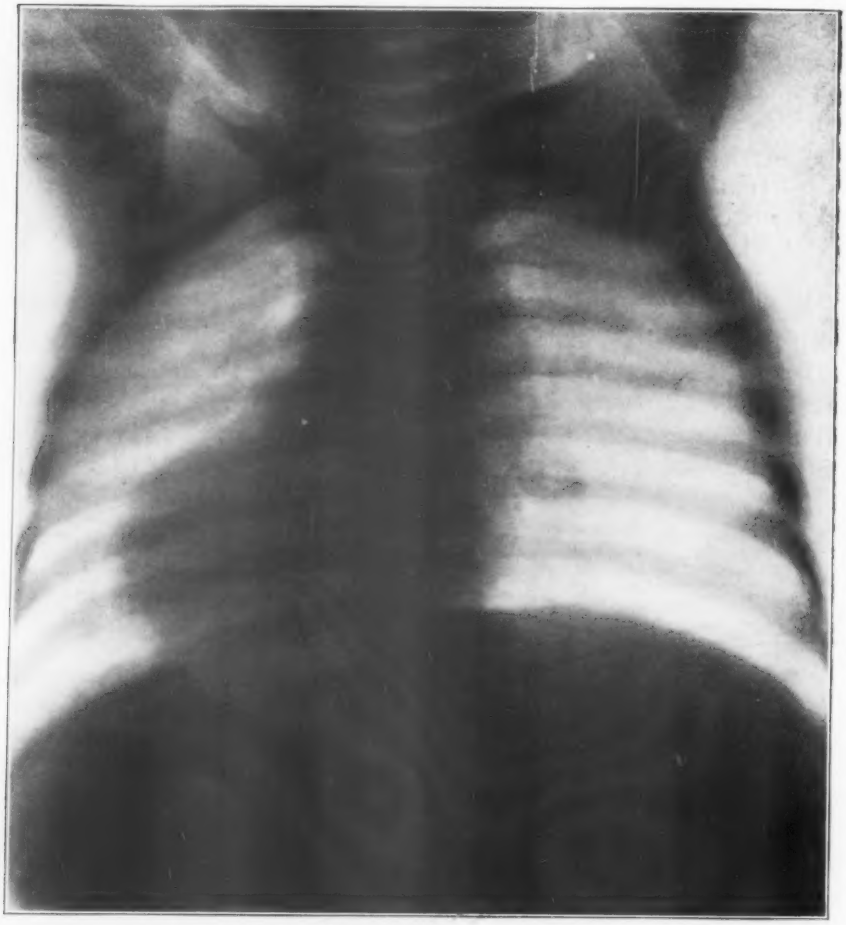
1. Hamburger, F.: Allg. Path. u. Diag. d. Kindertbc. (manuscript), 1910, p. 119.

2. Leiner and Spieler: Ergeb. d. inn. Med. u. Kinderh., 1911, p. 66.

In 1891, Barthelemy[3] first described the papulo-necrotic tuberculides, dividing them into two groups: Folloklis and Aknitis. The former involving only the epidermis, he thought might have some relation to tuberculosis; the latter, hard and subcutaneous, he claimed had no such relation.

In 1892, Pollitzer[4] called them Hydrosadenitis disseminata suppurativa sans destruans, because the histological examination in his case



Radiograph of thorax of L. M., Case 8, showing shadow at right hilus (bronchial gland) and infiltration of both apices.

demonstrated that the process was furthest advanced in and about the sweat glands. Later Dubreuilh,[5] Brocq,[6] etc., described similar lesions.

3. Barthelemy, J.: Ann. de derm. et de syph., 1891, p. 532.
4. Pollitzer, S.: Jour. Cut. Dis. incl. Syph., 1892, p. 9.
5. Dubreuilh: Arch. de méd. exper. et d'anat.-path., 1893, p. 63.
6. Brocq, L.: Ann. de derm. et de syph., 1897.

In 1896 the interest in the tuberculides was awakened, when Hallopeau, at the Third International Dermatological Congress first pointed out their relation to tuberculosis, and expressed the opinion that they were due to toxins produced by the tubercle bacilli from some deep-seated process. In the same year Darier[7] described the lesions very accurately, and first applied the name by which they are still known, i. e., *"Tuberculides cutanees."*

Boeck, 1898 and 1900, at the Fourth International Dermatological Congress, also expressed the opinion that they were due to tuberculotoxins acting on the vasomotor centers, giving for his reason for this hypothesis the symmetrical occurrence of the lesions. He also laid stress on the vascular and perivascular changes as previously described by Leredde. Boeck claimed that the spontaneous healing was another point in favor of the toxic theory. Philippson[8] (1898) described a case with thrombophlebitis of the deep veins of the subcutis. Török,[9] and Macleod and Ormsby[10] (1901) found similar changes. The latter authors demonstrated tubercle bacilli, thus strengthening the theory of bacillary origin. Jadassohn,[11] Haury and others, advanced the idea that they were due to attenuated or dead bacilli.

Pautrier[12] (1903) strengthened this latter view by demonstrating bacilli in sections, by producing tuberculosis in animals in whom excised tuberculides were placed subcutaneously, by positive tuberculin reactions and by finding tuberculosis of other organs.

Klingmuller[13] (1903) injected tuberculin, gotten by filtering crushed bacilli, into guinea-pigs subcutaneously, and by so doing produced typical tuberculides (Lichen scrofulosorum). These experiments greatly strengthened the toxic theory. Jadassohn[11] claimed that these results were due to fragments of bacilli in the emulsion. Whereupon Ziehler[14] (1910) got the same results with a filtered and dialyzed tuberculin.

Leiner and Spieller[15] (1908), through vaccination, brought about a typical eruption in guinea-pigs. Gougerot and Laroche[16] (1907-1909) produced typical lesions by rubbing virulent human bacilli into the shaven skin of guinea-pigs, as did also Lewandowski.[17]

7. Darier, J.: Ann. de derm. et de syph., 1896, p. 1431.
8. Philippson, L.: Arch. f. Derm. u. Syph., 1898, lv, 215.
9. Török, L.: Arch. f. Derm. u. Syph., 1901, lviii, 389.
10. MacLeod and Ormsby: Brit. Jour. Derm., 1901.
11. Jadassohn, J.: Handb. d. Hautk. (Mracek), 1907, p. 266.
12. Pautrier: Arch. f. Derm. u. Syph., 1904, lxix, 145.
13. Klingmuller: Arch. f. Derm. u. Syph., 1902, lx, 109; 1904, lxvii, 142.
14. Ziehler: Arch. f. Derm. u. Syph., 1910, cii, 37.
15. Leiner and Spieler: Verhandl. d. Gesselsch. f. Kinderh., 1908, xxvi, 115.
16. Gougerot and LaRoche: Arch. de méd. exper., 1908, p. 581.
17. Lewandowski: Arch. f. Derm. u. Syph., 1909, xcviii.

From 1909 on more and more authors were able to demonstrate the tubercle bacilli. Tileston[18] (1909) found bacilli in 70 per cent. of his cases; all of these being cases of miliary tuberculosis. Mathilda Lateiner[19] (1910) found bacilli in all tuberculides which she examined; six of her ten cases were miliary tuberculosis. Leiner and Spieler[15] were also able to demonstrate bacilli in a few of their cases, or to cause tuberculosis in guinea-pigs with excised lesions. Macleod and Ormsby,[10] Philippson,[8] Shaw and Laird,[20] etc., were also able to find bacilli.

From this short historical sketch it can be seen that although these so-called tuberculides were considered for a long period to be of toxic origin, all are now agreed that they are really of hematogenous bacillary origin.

PATHOLOGY

"The changes[2] in the epidermis vary according to the stage of the lesion; necrotic in the older, exudative in the younger. In the latter there is a pustule-like infiltrate between the horny layers of the skin, made up of leukocytes and degenerated epithelial cells, with a more or less far advanced necrosis in its center. Adjacent to this and following the course of the hair follicle, in the superficial layer of the corium there is a round, funicular infiltrate which shows a simple round-cell infiltration or one made up of lymphocytes, epithelioid-giant and connective-tissue-cells, with a central necrosis of varying grades of intensity. Within the necrotic area one frequently finds an obliterated or thrombosed vessel or the remains of such a vessel in the form of a ring of elastic fibers; and occasionally in or about this locality tubercle bacilli. There is, furthermore, edema of the deeper structures, and dilatation of the neighboring blood-vessels. Often a small-celled infiltration exists surrounding the sebaceous glands and hair follicles. Occasionally in the deeper parts of the cutis an obliterated or thrombosed vein is also found, or more frequently the walls of the latter show inflammatory changes. Frequently there are no typical tuberculous changes at all, but simply a non-characteristic piling up of cells in the upper layer of the corium."

SYMPTOMS

The tuberculides are very much more common than one is led to believe from the literature. They are so small and often occur in such small numbers, that, if one is not on the alert, and always looking for them, especially in infants, they will surely be overlooked. I have made it a practice in every case with a history of chronic cough, frequent "colds," loss of weight, pallor, intermittent, recurrent or continuous

18. Tileston: Arch. Int. Med., 1909, iv, 21.
19. Lateiner, Mathilda: Ztschr. f. Kinderh., 1910, ii, 442.
20. Shaw and Laird: Arch. Pediat., July, 1909.

fever, or where there is general glandular enlargement always to look for papulonecrotic tuberculides. When found I have always been able to make a positive diagnosis of tuberculosis, borne out either by later developments or by other symptoms and signs. If this is done regularly many more cases will be recognized before the disease has progressed to the advanced stage and one may by proper hygienic treatment reduce the mortality of tuberculosis in infants, provided that the case in question is not one of miliary tuberculosis. All of my cases, excepting one, a child of 16 months, have been in infants under 1 year of age. Leopold and Rosenstern[21] place the frequency of this sign in tuberculous infants at 40 per cent. Ten of their twelve cases were children under 1 year of age. That older children and adults also may have a similar eruption is evident from the case reports in the literature. They do not occur so frequently, however, nor are they of so great a prognostic significance in older individuals.

CASE REPORTS

CASE 1.—Child 4 months of age. Sent to one of the large European hospitals and treated there as a case of "Decomposition" (Marasmus).

Physical Examination.—A pale, greatly emaciated child (with skin hanging in folds all over the body, dry and inelastic, and no subcutaneous fat). On the inner side of the right thigh and the outer side of the left, in the diaper region, two small, reddish-brown, round spots, about 2 mm. in diameter, the one with a small scale on its surface, the other with a white central depression.

Chest: To the left of the spinal column, extending 3 cm. to the left of the level and just below the spine of the scapula, was an area of dulness and bronchovesicular breathing. No other abnormalities were found. The von Pirquet test was negative on two occasions.

In this case there was some question about the diagnosis even after I demonstrated the tuberculides. Those in charge thought the lesions too insignificant to be of any diagnostic importance. A few days later the child died and the autopsy revealed a general miliary tuberculosis.

CASE 2.—A. D., Italian child 6 months of age, came to Sydenham Hospital Dispensary May 31, 1911, for loss in weight, fever and ear discharge lasting three weeks, and a swelling behind the left ear since four days. F. H., aunt, living in the same house, died of tuberculosis one month before.

Physical Examination.—A pale, markedly emaciated child, skin hanging in folds, dry, inelastic. No rigidity of neck. No Kernig. Anterior fontanel admits three fingers; not bulging. Mastoiditis, with abscess formation on left side.

Chest: To the right of spinal column from second to fourth spinous process and 3 cm. to the right an area of dulness. Over the entire chest posteriorly, medium-sized moist râles.

Glands: Slight general enlargement.

Skin: There are six fading, brownish, shiny, flat papules 1 to 2 mm. in diameter, localized as follows: one each over right and left tibia, three on right thigh and one presenting the characteristic scale and central depression over the posterior aspect of the base of the first right metacarpal bone.

No other abnormalities; von Pirquet test negative; white blood-cells 7,200, polymorphonuclears 77 per cent., lymphocytes 21 per cent., eosin. 2 per cent.

June 1. Conservative mastoid operation performed (Dr. Jarecky).

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21. Leopold and Rosenstern: Jour. Am. Med. Assn., 1910, lv, 1723.

June 28. Child had been running a subfebrile temperature since operation. Wound clean but showed no tendency to heal. Sent to Bellevue Hospital, where a von Pirquet test done the following day was positive. Baby died July 9. No autopsy permitted.

CASE 3.—J. B., Italian, 11 months of age. Child brought to the dispensary Jan. 3, 1912, for cough, fever and loss in weight. Family history negative for tuberculosis.

Physical Examination.—Small, fairly well nourished child with evidences of slight loss in weight. Over entire chest and back, an uncountable number of pin-head to small pea-sized flat papules, very few of which show the characteristic scale or depression. They are of a pinkish to reddish-brown color. The mother said these had been present about five days. Scattered on the extremities were a few similar lesions.

Chest: At base of left lung posteriorly, dulness, bronchovesicular breathing accompanied by loud, crackling râles over an area about the size of a silver dollar.

Von Pirquet test very markedly positive. The mother did not return to the dispensary again, but about one month later I went to the home and found that most of the lesions had faded. Some, however, were still present and were of brownish, shiny appearance and flat. No changes in the chest signs. Evidences of the von Pirquet test still present.

May 27, 1912, I again visited the patient; the lung signs with the exception of slight dulness over the left base posteriorly had disappeared. There was a fresh crop of lesions on chest and back which had appeared one month previously, immediately after the child began to convalesce from measles. Before this, the mother said, the skin was perfectly clean.

January 21, 1913. Met the mother on the street, with the child. She said the eruption had entirely disappeared. The child appeared to be a fine, rosy-cheeked girl.

CASE 4.—S. I., Italian, 7 months of age. Brought to Sydenham Hospital Dispensary for cough and loss of weight.

Physical Examination.—Pale, emaciated child with skin hanging in folds on extremities.

Chest: At left angle of scapula a small area of dulness over which crackling râles and bronchial breathing are heard.

Skin: Over tibia on both sides, one 2 mm., brownish, flat papule with a whitish depressed center, shiny when the skin is stretched; von Pirquet test markedly positive.

Case then lost sight of.

CASE 5.—S. R., Italian, 16 months of age. Came to the eye clinic (Dr. R. Kahn) for phlyctenular ulcer of the cornea with a marginal keratitis. She was referred to me on account of cough and fever.

Physical Examination.—A pale, flabby child, considerably underweight.

Chest: At left base posteriorly, dulness, bronchial breathing and crackling râles. At right apex, slight dulness and harsh breathing; von Pirquet markedly positive.

Skin: Over left thigh about its middle, a 2 mm.-sized, brownish, flat papule with a central crust and depression.

Case lost sight of.

CASE 6.—T. M., Italian, 3 months of age. June 10, 1912, came to the dispensary for cough and fever lasting one week. Family history negative for tuberculosis.

Physical Examination.—Small, emaciated, pale child with cold extremities, mildly cyanotic. Skin hangs in folds and is inelastic. There is scarcely any subcutaneous fat. Slight rigidity of the neck.

Skin: There are five small, round, 2 mm. in diameter, livid to brownish, flat papules with a central whitish depression; two with a small scale. After the removal of these scales a small bleeding point is left at the base of the depression. They were situated as follows: three on the left thigh, outer aspect, one on the right thigh, inner aspect, and one on the back about over the right sacro-iliac junction.

Chest: In left anterior axillary line and extending over to the nipple line a small area of dulness over which there was bronchial breathing and crackling râles. Over the right apex posteriorly slight dulness. Otherwise no abnormalities; von Pirquet test negative.

Child died in hospital three days later. Temperature during this time ranged between 99 and 101 F.

Necropsy.—General miliary tuberculosis. A small area of consolidation was found at the outer, anterior border of the left lung; large caseating bronchial glands. Over the anterior surface of the right ventricle there was a miliary tubercle.

CASE 7.—Child seen at Mt. Sinai Dispensary, Dr. Bass' clinic. For the use of this case I wish to thank Dr. Bass.

S. K., 11 months of age, of Jewish parentage. Breast fed for two months only. Child is brought because she vomits once a day and is losing weight; has slight cough. Mother says rash on body has been present for six weeks.

Physical Examination.—A small, pale child, considerably underweight (12 pounds, 7 ounces). Skin has lost some of its elasticity. No glands palpable.

Skin: On the back about the level of the third lumbar vertebra are two small, flat papules, reddish-brown in color with a central, whitish depression, and a superficial scale. There is a similar spot over the head of the ulna which is more brownish and fading. Over the rest of the back there are several larger nodules, varying from about 4 mm. to 1 over the left shoulder, about 1 cm. in diameter. All of these nodules showed considerable infiltration of the surrounding tissues, especially the one on the shoulder, which was of a bluish-red color, very hard and had a very marked central necrosis and deep crater-like depression. This was evidently a very recent lesion. In its later course it became less infiltrated, had more of a brownish color and a typical central scale. Dr. Bass made the diagnosis of papulonecrotic tuberculides with which diagnosis I agreed. von Pirquet test seen twenty-four hours later was reported positive; when seen by us forty-eight hours after application appeared to be negative. The child in the next five days lost 3 ounces, but her condition was otherwise unchanged. She did not return to the dispensary again, but at the home I was informed that the child became very ill and was taken to the Babies' Hospital. For the use of the subsequent history and autopsy I take this opportunity of thanking Dr. L. E. Holt. She was admitted to Dr. Holt's service eleven days after she was last seen by us. The physical examination was about the same. White blood-cells, 7,600; polymorphonuclears, 56.6 per cent.; lymphocytes, 42 per cent.; eosins, 1.3 per cent. Blood culture was negative. Urine acetone+. Albumin +.

Jan. 5, 1913. Temperature rose to 103 F. and the lung signs were as follows: "In the region of the right nipple and toward the axilla is a small area the size of a silver dollar of dulness, distant bronchial breathing with an amphoric quality; von Pirquet negative."

January 11. Child had an attack of laryngeal stridor and swelling in the left side of vault of pharynx. Culture taken was negative for diphtheria. von Pirquet again done was doubtfully positive.

January 12. Lung signs show consolidation of right lower lobe.

January 13. A third von Pirquet was again doubtful.

January 14. Lung signs intense, frank pneumonia. White blood-cells, 8,000; polymorphonuclears, 69 per cent.; lymphocytes, 31 per cent. Stridor persists.

January 16. Child worse. Signs of consolidation over entire lower lobe and extending into the middle lobe where the above described (*vide* January 5)

small patch still persists, with distinct amphoric breathing. Child died at 12:30 a. m.

Necropsy.—Acute miliary tuberculosis with a patch of caseation in the right middle lobe. Large cheesy bronchial and mesenteric lymph-nodes.

CASE 8.—L. M., Italian, 6 months of age. Child brought for loss of weight, fever and cough which had lasted a few weeks. Family history negative for tuberculosis. Bottle fed.

Physical Examination.—March 9, 1912. Fairly well nourished, but pale child with evidences of slight loss of weight (12 pounds, 5 ounces).

Skin on left thigh one 2 mm. sized, brown, slightly raised, flat papule, with a white depressed center, on which rests an easily removable white crust.

Chest: Under the angle of the left scapula is an area about the size of a silver dollar of dulness, crackling râles and bronchovesicular breathing. These signs persisted about three months and then began to clear up gradually. In this time the child gained about 3 pounds. von Pirquet test markedly positive.

The child has been under my care for almost a year, reporting every ten to fourteen days. He has had frequent attacks of bronchitis and frequent rises in temperature, and is never entirely free from cough, but no tuberculides have appeared since the one first seen. He is fairly well nourished, now weighing 21 pounds at 17 months. He is, however, still pale, but walks around and were it not for his cough would be fairly normal. The chest signs, with the exception of an almost imperceptible amount of dulness over the sight where the old lesion was, are perfectly normal. von Pirquet test done one month ago was faintly positive.

Radiograph taken about one month ago by Dr. Joseph Friedmann shows a shadow at the right hilus (bronchial gland) and infiltration of both apices. This bronchial gland is probably the cause of his cough.

CASE 9.—J. M., Italian, 7 months of age. Feb. 11, 1913, the child was brought to Mt. Sinai Hospital Dispensary for cough and coryza for the previous three months. Occasional rises of temperature. The cough was hoarse, at times hacking, at times spasmodic, accompanied by mucus expectoration. No symptoms relative to the gastro-intestinal tract. Family history, one paternal uncle has tuberculosis.

Physical Examination.—Fairly well nourished, but pale child, with a fair amount of subcutaneous fat, but somewhat flabby.

Chest: An area of dulness posteriorly extending from second to fifth dorsal vertebra and to the right as far as midscapular region over which the breathing has a bronchovesicular character. Over the entire chest posteriorly medium sized moist râles are heard.

Glands: Slight general enlargement.

Skin: There are nine papulonecrotic tuberculides to be found, localized as follows: one on each cheek and one on the chin; these are flat, shiny, reddish-brown in appearance, the one on the chin having a small, shallow pin-point depression in its center; three on the back with the typical scale and depression; one on the chest, and two on the left thigh, outer aspect. von Pirquet reaction very markedly positive.

This case is still under observation.

PROGNOSIS

The prognosis, when tuberculides are present, is very bad. Sixty per cent. of Lateiner's,[19] 100 per cent. of Tileston's,[18] 40 per cent. of Leopold and Rosenstern's[21] patients died, and the great majority of these were miliary tuberculosis. Of my cases, four patients died, two are still living; two were lost sight of and one is still under observation, making a mor-

tality of 57 per cent. known. The other two patients probably also died, as they were in a very bad condition when seen. Of the living, the one child is in seemingly good health, strong and robust. The second is somewhat underweight and pale, but is otherwise normally developed, very active and playful, although he has a chronic cough and gets frequent attacks of bronchitis, and occasional rises of temperature lasting three or four days.

So that although the prognosis is generally bad, it is not an absolutely fatal one in tuberculosis under 1 year as claimed by Koplik,[22] Radziewjewski,[23] and others. These results correspond more with those of R. Pollak,[24] who had two cases, Ibrahim,[25] one case, and Bahrdt,[24] one case in which the process became latent.

DIAGNOSIS

Once having seen these lesions they will be easily recognized. The main thing is to look for them. They occur on any part of the body, but the most frequent locations are, on the thighs, especially in the napkin region, the extensor surfaces of the legs, forearms, dorsum of fingers and toes, and the lower half of the back. They may, however, occur on any part of the back, trunk and extremities, and occasionally on the face and lobule of the ear. For the most part the number of the lesions is very sparse, very often not more than two or three, but occasionally they are very diffuse and disseminated over the entire trunk and extremities (*vide,* Case 3). The v. Pirquet reaction is a great aid in clinching the diagnosis, especially in young infants. A negative reaction, however, in the presence of papulo-necrotic tuberculides should not be allowed to mislead one, as these lesions in infants frequently mean a general miliary tuberculosis, and it is known that in the latter condition the v. Pirquet and other skin reactions are frequently negative, especially in the last stages. The reaction may be of prognostic value, when tuberculides are present, inasmuch as those cases which reacted strongly, had a much better chance than those in whom the reaction was either negative or questionable or only slightly positive. Thus two of my cases that ended fatally had negative v. Pirquet reactions; a third was only doubtfully positive; and the fourth was negative once and slightly positive on a second occasion.

CONCLUSION

In conclusion, I may recapitulate that in nine cases of infantile tuberculosis, the diagnosis was first made by the finding of papulo-necrotic tuberculides.

22. Koplik, H.: Bull. Johns Hopkins Hosp., 1912, xxiii, 254.
23. Radziewjewski: Ztschr. f. Kinderh., 1911, ii, 560.
24. Hochsinger: (Quoted) Ztschr. f. Kinderh., 1912, iv, 293.
25. Ibrahim: Zeitr. z. Klin. d. Tuberc., 1912, xxi, p. 117.

These skin lesions are absolutely characteristic, and when once seen are not easily confused with other conditions. They are of extreme diagnostic importance, especially in infancy when the clinical diagnosis of tuberculosis is notoriously uncertain.

The majority of cases in infants are indicative of general miliary tuberculosis.

The prognosis is generally bad, but not always fatal.

The v. Pirquet reaction is a great aid in clinching the diagnosis. A negative reaction, however, must not be allowed to mislead one in the presence of papulo-necrotic tuberculides.

297 Central Park West.

THE PATHOGENESIS OF CASEIN CURDS IN THE STOOLS OF INFANTS*

ALFRED F. HESS, M.D.
NEW YORK

The subject of this paper is one that has interested pediatrists both in this country and in Germany for many years, but was brought forth anew for discussion a few years ago. It is hardly necessary to review the history of investigations in this field. The articles have been many, and almost all have included a summary of the numerous investigations which have considered the problem of casein digestion. It is well known that although Biedert regarded protein indigestion as the main obstacle to successful artificial feeding of infants, this conception has been completely disavowed by his fellow countrymen. The question was not allowed, however, to slumber long, but was aroused again by Talbot,[1] who described large, hard masses in the stool, which he termed casein curds, believing them to be composed mainly of the casein of the milk. Without enumerating the various writers who immediately took sides on this important question, it should be said that Meyer and Leopold,[2] Southworth and Schloss,[3] Ibrahim,[4] Brennemann,[5] and Courtney,[6] published investigations for or against this partial retrogression to the old Biedert idea. As the result of intensive work on this subject, advance was rapidly made. Although no attempt will be made to detail the various experimental studies in this field, some few must be mentioned. In the first place, it was definitely shown by Talbot by means of various biologic tests that these curds do contain casein. It was, however, impossible by means of sensitive precipitin and hemolytic reactions to estimate how great a part of their substance the casein constituted. Another advance, shedding light on this subject, was the almost simultaneous reports of Ibrahim and of Brennemann showing that boiling the milk prevents the appearance of these masses in the stool. These studies, however, have not entirely elucidated the question. It is argued that these bean-like masses are only in part, perhaps in small part, composed of casein, and that although protein in nature, the main protein substance consists of

*From the Research Laboratory, Department of Health, New York City.
*Read before the New England Pediatric Society, March 1, 1913.

1. Talbot, F.: Arch. Pediat., December, 1909; June, 1910.
2. Meyer, L. F. and Leopold, J.: Arch. Pediat., October, 1909; February, 1910.
3. Southworth, T. and Schloss, O.: Arch. Pediat., April, 1909.
4. Ibrahim, J.: Monatschr. f. Kinderh., 1911, x, 55.
5. Brennemann, J.: Am. Jour. Dis. Child., May, 1911.
6. Courtney, A. M.: Am. Jour. Dis. Child., January, 1912.

intestinal secretion, which is mingled with the fat of the milk. Furthermore, they do not show whether the curds are formed in the stomach or in the intestine, or what leads to their formation, or how it is that in spite of the proteolytic enzymes, they pass through the entire intestinal tract undigested.

PLACE OF FORMATION OF CURDS

In the course of the care of a large number of infants in an asylum, this question was brought to notice, and has been a source of interest and consideration for the past year or more. It seemed almost certain that these masses in the stool are similar to those which are found in the stomach contents or in the vomitus of infants, but the difficulty lay, as is often the case, in finding a means of proving a palpable fact. In this connection, we wish to emphasize a point which we feel has not been granted sufficient importance, one which all investigators have noted, namely, that the interior of these masses is always white, although the exterior is stained brown or green by the stool. This fact would seem to afford very strong proof that these masses are formed in the stomach. For if they were formed in the small intestine beyond the point where the bile enters the duodenum, they undoubtedly would be mixed with bile, and their interior would be bile-stained. From this view-point, the bile may be regarded merely as a colored fluid which is injected into the upper duodenum, just beyond the pylorus. Nature provides in this way a most simple and convincing experiment, one which might well be devised were we to resort to animal experimentation for our purposes. Unformed substances which come from the stomach will certainly be mixed and tinged with the bile, whereas formed substances may remain unmixed and uncolored. If this be granted, it does not seem reasonable to dodge the conclusion that these masses must be formed before they enter the upper duodenum.

Another fact closely allied to the former and pointing in the same direction, is that the main fatty constituent of these masses is not fatty acid, but neutral fat; that the curd has not been acted on by lipase which enters at about the same point as the bile. Neither the bile nor the pancreatic fluid, therefore, has access to the interior of these masses.

A new method of approach to the question of the gastric or intestinal formation of these stool curds was afforded by the employment of the duodenal catheter. Last spring there was an infant, 3 months of age, in my hospital ward, who quite regularly passed casein curds. Its food consisted of half milk and half barley water with the addition of 5 per cent. cane-sugar; of this mixture, 6 ounces were given every four hours. For the purpose of preliminary experiment, this infant was fed solely on barley water for forty-eight hours, during which time the stools became brown and scanty, and the casein masses disappeared. It was then given

by means of the duodenal catheter the same milk mixture which it had received before, and was fed very slowly five times in twenty-four hours. Duodenal alimentation was thus continued for forty-eight hours and the stools became yellow and larger, but contained no bean-shaped masses. Ordinary bottle feeding was again instituted, for the first two days merely barley water, and then the same milk mixture as originally given. Forty-eight hours after the milk was given by mouth, the casein masses reappeared in the stool. This would seem to show that these curds are formed in the stomach and disappear if the food is made to circumvent the stomach. The bile coloring test mentioned above, as well as the duodenal feeding test seem conclusive on this point.

INHIBITING CURD FORMATION

A series of experiments were undertaken with the view of inhibiting the formation of the casein masses. Many of these methods had been resorted to previously by others. For instance, boiling the milk (in these tests for five minutes) caused the masses to disappear. We were also able to confirm the results of others who found that 2 grains of sodium bicarbonate or of sodium citrate in an ounce of milk did away with their formation. Calcium chlorid, in 10 per cent. solution, added in the ratio of 1 to 20 to the milk was most effective, although not invariably so, in doing away with the formation of the curd masses. It was, however, not so easy to produce the curd at will; the most successful method was to give rennin at the time of each feeding. In many cases, but by no means in all, this was followed by the appearance of the large curds in the stool. Diluting whole milk with one-third barley water does not prevent or materially decrease the formation of the curds. We have had very many babies on this formula who passed casein curds, indeed, the most marked instance of curd masses which we have met with, occurred in the case of an infant who was receiving a mixture of this kind.

The question of the effect of heating the milk was entered on in somewhat greater detail. Milk was heated to varying temperature for successive days and it was found that when the milk was brought to about 170 F. the curds almost always disappeared. They then occurred only occasionally and the appearance of the stools in this regard was quite different from that when milk was given that had been heated to a less high degree. We were especially anxious to determine the effect of pasteurization at 147 F. for forty-five minutes, because this is the type of pasteurization which is being employed by various dairies, and fully complies with the latest ordinance of the city of New York. Last year for this purpose some milk was pasteurized to this degree in our own apparatus, and this year milk was purchased which was thus pasteurized by the holding process at the creamery. It was found that pasteurization

at this temperature did not do away to an appreciable extent with the formation of the curds in the stools; indeed, when recently in the course of experiments bearing on this question, we put fifteen infants on raw milk, we did not notice any increase in the formation of casein curds. In this connection, it may be added that an experiment was made to see the effect of freezing the milk. It was found that the curds appeared just the same, even after milk was given which had been frozen solid for a couple of hours. The substance, therefore, which goes to form these curds acts chemically like casein, which is changed in its power of coagulability by a temperature of about 170 F.

STUDY OF STOMACH CURDS

Having assured ourselves that these curds were formed in the stomach, we directed our attention to the gastric digestion. With this end in view we used the stomach tube and evacuated the stomachs of many children, some of whom frequently passed curds, and others who were normal in this respect. However, we met with the same unsatisfactory experience as Tobler,[7] who found that he was unable by this means completely to empty the stomach, and proved by means of the radiograph that some milk always remains behind. In our endeavors to overcome this difficulty, we used a large stomach tube; we irritated the pharynx with the tube or with the finger; we gave syrup of ipecac, and at times followed this by warm water given by the tube, but were unable by any of these means to incite vomiting at will. This was quite surprising in view of the marked tendency of infants to vomit at the least digestive or febrile disturbance. Indeed, we found that even when the infant vomits, the stomach does not always completely empty itself, as was shown by the fact that a second or even a third attack of vomiting resulted in the renewed evacuation of large curds. So that at present it is impossible to carry out a satisfactory study of the gastric curds of infants. However, we examined the curds which we obtained by means of introducing the tube, as well as those from time to time vomited spontaneously. On comparing the size and the firmness of the curds of those infants who show casein masses in the stools with those who do not, it is evident that the former are larger and firmer. They are nevertheless always softer than the curds in the stool. But we have merely to shape these curds from the stomach, and to expose them for some hours to the air, to render them brown and at times almost indistinguishable from the bean-shaped curds of the stool. They are not so firm as the latter, probably because they have not undergone the continued pressure of the intestinal peristalsis. Apart from the difference in curd formation, nothing distinctive was noted in regard to the gastric contents of infants having casein

7. Tobler, L.: Ergeb d. inn. Med., 1908, i.

masses in the stool. The stomach contents was not exceptionally acid at any stage of digestion. This is what we should expect when we consider that rennin is inhibited by a high degree of acidity. Free hydrochloric acid was occasionally met with.

It may be of interest to add a word concerning some attempts, although they proved negative, to increase the size or firmness of the gastric curd. Tobler[7] states that the curd can be kept for long periods in the stomach if the infant is fed very frequently, so that the solid matter does not gain an opportunity of passing the pylorus. This idea was carried out on three children who were fed every hour with about three to four ounces of milk. Although they had at some previous time passed curds, during this three days' test no curds were found in the stool.

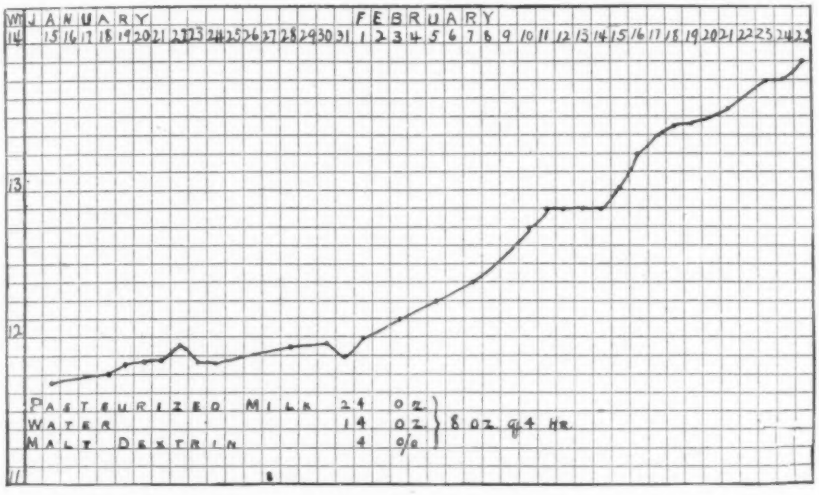

Chart showing steady gain in weight in spite of daily passage of large curds. I. L., aged 6 months.

Infants fed by the cup did not react differently from those fed by the bottle, nor did those fed rapidly through a large opening in the nipple, differ in their stools from those in whom the food was given slowly through a small aperture.

STUDY OF DUODENAL CONTENTS

So much for the formation of gastric curds. We next proceded to study the duodenal contents of these children. Many hundreds of examinations by means of the duodenal catheter had given us a definite idea of the curds normally found in the duodenum in infants. These curds are typical, to such an extent indeed, that they have been of aid to us in judging whether the tube has passed the pylorus; they may be regurgitated from the duodenum into the stomach. We have always noted them

in our records as *pyloric casts.* They vary in length from about ⅛ to ½ an inch, and give the appearance of having been compressed through a narrow channel. They are quite what we should expect when we reflect on the radiographic pictures of milk in its passage through the pylorus.[8] These curds afford us an opportunity of judging of the size and nature of the curd which normally enters the duodenum when an infant is fed on cow's milk. The duodenal contents of infants who passed the casein masses, especially those who passed these bodies constantly and in large numbers, was frequently found to be different. Although the diameter of the catheter prevented our obtaining a true picture of the size of the curds, it was noted that in these cases larger and coarser curds were aspirated, and that the lumen of the catheter became frequently plugged.

An examination of the trypsin was carried out in these cases. As the first step, the duodenal contents were aspirated from some infants who passed curds. It was found that the trypsin in these cases was normal, and was able to digest gelatin within twenty-four hours. It was just as potent as in infants who were not passing curds. Second, the curds from the stools were subjected to the action of the trypsin of the duodenal fluid of normal children, and it was found that even after they were kept under these conditions over twenty-four hours in the thermostat, the firm masses were not completely digested. In view of the fact that trypsin is one of the most powerful of the proteolytic ferments and readily digests casein, these tests show the importance of the density or compactness of a substance quite apart from its chemical constitution, in respect to its digestibility, and in particular of these casein masses. It should be considered, however, that these masses are probably much firmer when they are passed, than throughout their course in the small intestine.

A further experiment was instituted as a corrollary to these tests of trypsin. The curds of the stool were scraped, thoroughly washed with water, and then fed to some infants who passed curds, and to others who did not have them in the stools. Before feeding them, they were injected in the interior with fuchsin, so as to facilitate their recognition later. These curds were recovered in the stools from normal babies who for some time had not had casein masses in the stool. All of them were not found, but enough were recovered to show that these masses are incapable

8. These pyloric casts are frequently enveloped in a coat of mucus, which can be readily seen if the cast is floated on water, or in a fluid such as toluol which makes the enveloping substance more opaque. When this is done, we frequently find a long tail of mucoid substance attached to the curd. These bodies may be similar to the food strings noted in the duodenum by Cannon by means of the fluoroscope. They are not formed merely in the passage through the catheter, as they cannot be obtained from the milk within the stomach, and are frequently of a diameter smaller than the catheter.

of being digested in the gastro-intestinal tract of the normal infant. Similar experiments with the gastric curds were undertaken, but thus far are not conclusive.

OCCURRENCE OF CURDS AND EFFECT ON HEALTH

Our series of investigations may be divided into two groups, both as regards time and method of approach. The first one taken a year ago consisted in noting throughout the asylum the various cases in which curds were found in the stool, and in keeping for months a daily record of these cases. This showed us that there were certain infants who passed these curds for long periods, although they seemed to be in perfect health, and no wise differing from others who were obtaining the same food and whose stools were normal.

More recently we have undertaken an experiment less broad, but more intensive in nature. We inspected the stools in a ward composed of sixteen infants, ten of whom were obtaining 40 ounces of ⅔ milk with 5 per cent. sugar, and who varied in age from 6 months to a year, and six infants varying in age from 1 to 2 years, who were receiving a pint of milk, together with mixed diet. The milk had been pasteurized at 147 F. for forty-minutes. For a period of over a month, every stool was saved and inspected either by the house physician or by myself, and a record made as to its character and whether or not it contained casein masses. Vegetables were not given, as their coloring matter interfered with the examination of the stool. In the course of this month there was no child that did not at some time pass casein masses. Some passed them far more regularly than others; the younger group passed them more frequently than the older group. There was the same individuality noticed as had been remarked on last year; certain infants, independent of their well-being or of any clinical manifestation, had a peculiar tendency to pass bean-like masses in their stools. This tendency seemed to persist, for on examining the stools of the infants who passed curds so frequently last year, and who now are in the high-chair ward, it was found that many of them, an undue proportion, are still passing curds. The curds are not so large nor so hard in any instance as they were when the infant was younger. Incidentally, it may be of interest to note that in the course of these examinations of all the stools, it was most striking that certain infants have characteristic stools apart from the presence of curds, and that frequently the observer is able to designate the stool of a certain child by its appearance and odor, although the other children are on the same diet. It was also soon evident that it is impossible to draw a sharp distinction between casein and non-casein material, for there are gradations in the curds. Some, although small, soft, and easily broken up, are clearly, judging from their appearance, of the same constitution as the

firmer and larger masses, and differ solely in having undergone partial digestion.

There is one more question which should be considered, namely, whether this disturbance is purely gastric in nature, or whether it is also dependent on some intestinal derangement. We have seen that in these cases there is no deficiency of trypsin. However, a lack of erepsin, the other intestinal ferment which is able to digest casein, would also favor the non-digestion of these curds and lead to their passage in the stool. The same would be true of any condition which increased intestinal peristalsis, although the secretion of ferments was normal. It can be definitely stated that these casein masses are found not only in normal stools, but in the stools of infants which have been normal for many consecutive days. They do not, therefore, viewed from the clinical standpoint, indicate an intestinal disturbance, nor do they by mechanical irritation lead to intestinal trouble. However, although there is no parallelism in this regard, when we look over the notes of about one thousand stools of the group of sixteen infants who were continuously observed, it is clear that the curds are found more often in stools that are not completely digested, and at times when there is some faulty digestion, than when the intestinal functions appear normal. They are noted most often when an infant had three or more stools in the course of twenty-four hours, but were not infrequently present when there was but one or two stools in the course of the day. Judged by the various clinical criteria, they seem to indicate simply non-digestion of abnormally curded casein, and not a specific type of indigestion,[9] for there may be a steady gain in weight and no signs of indigestion, no loss of appetite, no vomiting, no rise of temperature. It is significant in this regard that the infant which gained the most weight and with the greatest regularity during the month of observation, chanced to be the very one which had curds most regularly in the stools (Chart 1).

CONCLUSIONS

From what has been stated, it would seem certain that the large bean-like masses in the stool concerning which there has been such difference of opinion, are curds which contain casein, and are formed in the stomach. The fact that they show no bile coloring matter in their interior goes to prove that they are moulded before they reach the level of the duodenum where the bile is poured into the intestine; the

9. It was found that when 1 per cent. of powdered casein is added to the daily food of infants who pass these large curds, it does not result in the appearance in the stools of a greater number of the casein masses. If the disturbance were due simply to a lack of casein digestion or a casein indigestion, we should expect an increase of the bean-like masses. Viewed from the intestinal standpoint it is rather a casein-curd non-digestion. We cannot consider the digestive disturbance a "raw milk dyspepsia" as Ibrahim has suggested, as it occurs so frequently when pasteurized milk is given.

additional fact that they disappear when the food is introduced by catheter directly into the duodenum, and reappear when it is once more given by mouth, must be accepted as strong evidence towards this conclusion. We wish to lay emphasis on the observation that certain infants are peculiar in their tendency to pass the curds with slight intermissions for months, whereas other infants similarly fed pass stools normal in this respect. This shows that in studying the pathogenesis of this condition we must take a clinical as well as a laboratory point of view, and must consider the individual infant and not merely the chemical factors which favor or hinder the coagulation of milk in the stomach. There are some infants who produce larger and firmer curds in their stomachs, due either to the amount of rennin which they secrete, or to the abnormal conditions under which the enzyme acts on the milk. These larger masses were found in the stomach and also in the duodenum of infants who passed casein curds in the stools. Ordinarily, the milk curd is pressed from the pyloric curd of the stomach into the duodenum in the form of slender, compact "food strings," which may be termed *pyloric casts*. In the cases with curds in the stools, the catheter showed the duodenum to contain larger masses.

The curds are always of gastric origin and their pathogenesis is always gastric. The intestines play merely a secondary rôle. From the intestinal standpoint the curds indicate merely the non-digestion of casein masses. The intestinal disturbance which at times accompanies the appearance of these masses in the stool, may be of gastric orgin, or of the same origin as the disturbed gastric function; it is not caused by the curds, nor is it the cause of the curd formation.

We cannot close without considering whether this clinical condition is harmful, and whether therefore, therapeutic measures should be taken to obviate the formation of these curds. Although a healthy infant may thrive in spite of the curds, we cannot infer that they may not sometimes lead to more serious disturbance. Furthermore, it is open to question whether foreign bodies of this size should be allowed to form and to traverse the intestinal tract. Diluting milk with barley water does not check their formation, nor does pasteurization to 147 F. for forty-five minutes, a process which complies with the ordinances of the health departments of various cities. Pasteurization to 170 or 175 F. causes the curds to disappear to a large extent, and therefore may be said to render the milk more digestible in these cases. We believe that the curds should not be allowed to persist, but should be checked, either by a high degree of pasteurization or by boiling the milk.

154 West Seventy-Second Street.

THE DIAGNOSTIC VALUE OF THE v. PIRQUET TEST

SECOND PAPER

F. L. WACHENHEIM, M.D.
NEW YORK

In July, 1912, I published a paper,[1] in which I tried to show the positive as well as negative value of the cutaneous tuberculin test in both young and older children. Since that time I have succeeded in collecting new material, fifty additional cases, making a total of 100. I hereby present an analysis and summary of both series, going into details as far as possible, especially as regards consideration of the ages of tested patients.

I again begin by excluding the clinically tuberculous, thirteen in the first series, seven in the second, leaving, respectively, thirty-seven and forty-three children to be considered. I need hardly say that the excluded cases invariably gave a positive reaction.

All the remaining eighty children were *suspected* of tuberculosis, because of a chronic cough, marked impairment of general nutrition, enlarged lymph-nodes, or some other of the signs commonly called scrofulous. They therefore represent a material selected for its likelihood to react positively.

I shall begin by dividing the total into three age groups, namely, under 6, 6 to 9 and 10 or over. The resulting classification follows:

	Under 6	6 to 9	10 and over
First series	14	14	9
Second series	10	22	11
Total	24	36	20

In the first age group the number of positive reactions was 0 in both series; at this age the test is, therefore, almost infallible, and trustworthy even beyond the favorable view usually taken. We may regard it as proven, that, after excluding miliary tuberculosis and a few acute febrile diseases, notably measles, the cutaneous reaction may be depended on from both points of view.

In the second age group the respective positive results were 0 and 2 in number; percentages, 0.0 and 9.1; total percentage, 5.6. In the third age group the numbers were 2 and 1; percentages, 22.2 and 9.1; total percentage, 15.0. Percentages in children of 6 years and over in the two series, were, respectively, 8.7 and 9.1; average, 8.9. The two series agree

1. Am. Jour. Dis. Child., July, 1912.

so closely, when summarized, that they evidently represent accurately the general run of cases in my dispensary service.

All of these figures are lower than those recorded by the majority of other observers; furthermore, they are based on a selected material, from which robust children have been carefully excluded. There is, therefore, no doubt that, at least on the East Side of Manhattan, most of the children that look tuberculous are free from that disease; their condition is due to other and less serious causes. On the other hand, and just because of the above facts, a positive reaction should, as I stated in my first paper, be regarded with the utmost seriousness. There is no scientific reason to assume that the tuberculous infection is obsolete or even arrested. These children are in any case fit subjects for treatment in a preventorium.

For some reason, not easy to comprehend, the results in my first paper were criticized for referring to selected material, though we should expect that a test of miscellaneous children would give even lower figures. As a matter of curiosity, I have also tested a series of fifty consecutive children, aged 6 years or over, excluding such as were probably tuberculous. Of these, five gave a positive reaction, 10 per cent., a proportion practically identical with that found in run-down children. Let it not, however, be assumed that this last group included markedly healthier children than the others; there were no really normal children among them, for normal children are not brought to the dispensary. It is notorious that the older children of the poorest classes are nearly all subnormal in some respect, and this is certainly true of dispensary patients; it is therefore natural that this last group be in all respects similar to the others.

SUMMARY

I may summarize as follows: More recent investigations accurately bear out my earlier statements as to the actual prevalence of tuberculosis among the poor children of the upper East Side of New York, referring especially to those of Jewish extraction, being probably less applicable to those of Italian origin. I repeat that the very smallness of the ratio of positive reactions emphasizes their clinical and prognostic importance. We do well to act accordingly, and treat the affected patients as at least potentially the victims of a serious tuberculous lesion.

111 West Eighty-Fifth Street.

SEVENTIETH ANNIVERSARY OF ADOLPH BAGINSKY

Adolph Baginsky was born in Ratibor, Prussia, May 22, 1843. He studied in Berlin from 1861 to 1863 and later was a student of Virchow and Traube. He studied in Vienna in 1864, and again in Berlin, in 1865 and 1866. During an epidemic of cholera in 1866 he was an assistant in the city hospital. He passed his state board examination in 1867, and entered the practice of medicine in 1868 in the town of Seehausen near Magdeburg; he was in charge of a military hospital in 1870 and 1871; in 1872 he returned to Berlin where he founded a polyclinic for children's diseases which he continued for many years.

In 1896, in conjunction with Rudolph Virchow, the Emperor and Empress Frederick Children's Hospital was established, with Baginsky as the Director and Supervising Physician. This institution was endowed by the Empress Frederick to commemorate the death of her husband. The institution has increased in size and covers a large area and has now been acquired by the City of Berlin. There are large buildings devoted to scarlet fever, diphtheria, whooping-cough, measles, typhoid fever, vulvovaginitis and syphilis; all internal diseases are admitted to a large separate building devoted to acute diseases. A correspondingly large surgical pavilion, an orthopedic department with x-ray and pathological laboratories, a nurslings building for the study of metabolism, with a chemical laboratory supervised by Dr. Sommerfeld comprise another group of buildings.

All deaths occurring in the German hospitals are autopsied, hence the great value of clinical, bacteriological, and pathological laboratories.

In 1877 Baginsky in conjunction with Alois Monti founded the *"Centralzeitung"* which two years later was changed into the *Archives of Children's Diseases*, of which Baginsky is still chief editor. About four volumes have been issued thus far. Amongst the many scientific contributions of Baginsky may be mentioned his Manual of School Hygiene, appearing in 1876. Since then many editions have been printed. A text-book on Diseases of Infancy and Childhood has seen almost a dozen editions. His book on Serum Therapy in Diphtheria appeared in Berlin in 1895. A book on diphtheria and diphtherial croup was part of Nothnagel's Manual of Special Pathology and Therapy.

In 1891 Baginsky was appointed an Extraordinary Professor of Diseases of Children in the Berlin University, which appointment he still holds. In 1890 he edited a *Festschrift* in honor of the seventieth birthday of Professor Henoch. Although many articles and scientific contributions have been published on the treatment of diphtheria by antitoxin, none has proven more valuable than Baginsky's, published in 1894.

Baginsky has always been in the front rank of those who have fought for progressive hygiene in children, and his research work and work in physiological chemistry are too well known to require repetition.

Baginsky was married to Clara Rosenberg who died about fifteen years ago. He has one son, Dr. Arnold Baginsky, at present connected with a city hospital of Berlin. His American colleagues unite in congratulating him on his seventieth anniversary (May 22d) and in wishing him continued health so that his scientific work may be continued for many years.

NEW YORK. LOUIS FISCHER, M.D.

PROGRESS IN PEDIATRICS

RÉSUMÉ OF THE LITERATURE ON INFANT FEEDING AND DISEASES OF THE DIGESTIVE SYSTEM, FROM JULY, 1912, TO MARCH, 1913

MAURICE OSTHEIMER, M.D.
PHILADELPHIA

INFANT FEEDING

BREAST FEEDING

The importance of breast milk is the main topic of medical discussion in infant feeding. This is noticed in many articles.[1, 6] Herrman[1] observed 139 babies for a year after birth. Eighty per cent. were breast-fed. Though six were premature, only 14 out of 139 died; a mortality under 10 per cent. Although one mother believed that her milk-supply was scanty, regular weighing showed steady gains in weight. Another baby, weaned because the mother left for operation, was later returned to the breast successfully. Griffith[6] concludes that the ability of mothers to nurse their offspring is no longer materially decreasing; that conditions are improving among the well-to-do and not changing for the worse among the poor; and that the infrequency of nursing depends chiefly on the ignorance of the laity and the indifference of the physician. If the mother has not enough breast milk at first, Von Reuss[7] advises using milk from another's breasts. Crookshank,[8] who lays emphasis on the biological qualities of the food given the mothers, advises a wet-nurse as the best substitute for the mother. Sedgwick[9] shows that about 80 per cent. of the wives of American physicians succeed in nursing their children three months or longer. Davis[10] has collected a mass of mortality statistics to show the value of breast feeding. Risel and Schmitz[11] observed 824 infants during 1910, to whose mothers the city of Leipsic paid nursing premiums. No child who was nursed for three-fourths of its first year died during the first year. By test-feeding, Pritchard[12] shows that the amount of breast milk taken by infants is far below our usually accepted estimates. He, as well as Comroe,[13] Morse,[14] Brady[15] and Ostheimer,[5] advocates three-hour feedings, provided nutrition is well maintained. The advantages of longer feedings, according to Comroe,[13] are absence of indigestion, regular gain in weight, gain in appetite, regular sleep and more time for the mother. Von Reuss[7] advises only five or six feedings in twenty-four hours. Brady[16] states that breast milk is most effective in raising the food tolerance, especially in summer diarrhea.

The difficulties of breast feeding are discussed by Beer.[17] A baby may be too lazy to nurse. It is also common for neurotic women to suffer from intermittent muscular contraction in the breasts, causing periodic diminution of the milk-supply. In such cases a wet-nurse is of value, or supplemental feeding, as advised by McClanahan,[18] should be begun. Von Reuss[7] advises beginning supplemental feeding when the baby reaches 6 months and weaning totally at 9 months. Decherd[19] begins to give the breast-fed baby one bottle daily on the eighth day of life; weaning is then possible, should it become necessary. Most physicians will object to this. Lust's[20] examinations of the breast milk from twenty-five women showed a milk sugar content of from 5.7 to 8.5 per cent. Schmid[21] noticed dyspepsia in infants under 3 weeks of age when the mothers got about too early, because babies were fed every time they cried. For the baby's sake, therefore, the mother should be kept at rest for two weeks after delivery. Stolte[22] noted, in infants who have lost much weight, that breast milk, even if given when the baby is recovering, may produce still further loss of weight. He considers it important to make the baby take enough breast milk to prevent further loss of weight. This amount, however, may cause diarrhea. Knox[23] found that babies of the working class could be kept well during summer heat of moderate intensity if their rights to mother's milk were not denied them. It is interesting to note that Peiser[24] was able to preserve the superfluous breast milk of wet-nurses with hydrogen peroxid, keeping it on ice, unspoiled, for as long as fifty-two days. Soxhlet[25] and Langstein and Edelstein[26] have investigated the iron content of human and cow's milk. They find in cow's milk on an average only one-third as much iron as in human milk, often only one-fifth and rarely somewhat over one-half. Therefore, artificially-fed infants may show pallor from the want of iron.

ARTIFICIAL FEEDING

CERTIFIED MILK

Certified milk is coming into general use. Tenney[27] speaks of the advantages of certified milk and the need for a Milk Commission in every city. Coit[28, 29] and Van Derslice[30] also extol certified milk. Coit[31] tells the standards of the American Association of Medical Milk Commissions; Newton[32] discusses recent hygienic improvements in dairying; Nance[33] and Gamble[34] emphasize the value of "score-cards" in marking conditions at the farm; while Bell[35] describes the New York Sanitary Milk Dealers Association for the improvement of the milk-supply and the observance of state and municipal laws and regulations. This is a great advance over the conditions which existed formerly. Schorer[36] discovered that ordinary milk has more dirt, but fewer cells than certified milk. Rosenow[37] found, however, that certified milk may contain virulent streptococci and pneumococci, when epidemic sore-throat prevailed.

PASTEURIZED MILK

Jordan's article[38] is a strong defense of pasteurization. Morse[39, 40] advises pasteurizing all except the cleanest milk. He states that it is impossible to determine whether babies fed continuously on pasteurized milk thrive better than those fed on raw milk, or whether the former are more apt to develop the diseases of nutrition. To pasteurize, he keeps the milk, divided into separate bottles, at 145 F. for 30 minutes. Winslow[41] states that the only way to overcome the possibility of tuberculosis in milk is to pasteurize it; and the only safe pasteurized milk is that prepared at home. Rosenow[37] advises pasteurizing even certified milk when epidemic sore-throat is prevalent. Kilbourne[42] has described a device for controlling the temperature to which the milk is heated during pasteurization.

BOILED MILK

Brennemann[43] continues advocating the use of boiled milk. There is no evidence to show that babies thrive less on boiled milk than on raw cow's milk. All foreign reports give good results. The hard, tough curds formed in the stools of infants fed on raw milk, with diarrhea and dyspepsia, disappear when boiled milk is given. The casein of raw milk, unless modified so that it will not form hard, large curds, offers serious difficulties in digestion that are not present in boiled milk. Plantenga[44] observed that infants fed on raw or boiled milk for a prolonged period showed more watery stools while on the raw milk. Milk boiled and utilized at once does not produce scurvy; but if boiled and kept over twenty-four hours, the milk probably will cause scurvy. Morse[40] advises boiling all except certified milk, in spite of the fact that cooking makes the milk somewhat less digestible and that its continued use predisposes to the development of the diseases of nutrition. Coit,[45] who reviewed the latest reports on boiled milk, concludes that the boiled milk of a foreign species gives better results than the same milk raw.

METHODS OF FEEDING

Fischer[46] advises common sense in feeding infants, avoiding all fads and studying the individual baby. He begins with diluted whole milk, one in five, with 5 per cent. maltose added. Then he increases gradually. Ostheimer[5] advises beginning with whole milk diluted one in four, then one and a half in four, then half and half; then using the upper 24 ounces off the quart, half and half, then the upper 20 ounces off a quart, half and half. By this method, in the vast majority of instances, feeding has been successful. Torian[47] believes that the young infant should not ordinarily be fed on whole milk mixtures. Pritchard[12] advises beginning artificial feeding on low formulas. He even predigests the mixture at first. Torian[47] says that the calorie value of every mixture should be

calculated. To use both the caloric and percentage methods is the advice of Morse,[14] and Everhard and Felker.[4] Amos[48] quotes the rules formulated by Cheadle. Scott[49] feeds according to the age of the infant.

Ladd[50] has described a short method for calculating percentage modifications, using 16 per cent. fat cream and fat-free milk. He divides the fat percentage desired for a 20-ounce mixture by 0.8, which will give the number of ounces of 16 per cent. fat cream. He multiplies this number by 0.15, subtracts the result from the protein percentage desired and divides by 0.2, which will give the number of ounces of fat-free milk. He divides the sum of ounces of both cream and milk by 4, subtracts the result from the sugar percentage desired and divides by 2, which will give the number of level tablespoonfuls of sugar to be added. To calculate the diluent, he subtracts the sum of the cream and milk from the 20 ounces. Graphic feeding charts, one more complex than the other, are described by Smith[51] and Cowie.[52] Torian[47] considers it impossible to feed infants intelligently unless gross examination of the stools is made daily.

Much emphasis has recently been laid on educating mothers and young girls in hygiene and infant feeding. Cooley[53] considers that this education should be directed by the pediatrist. Knox[23] and Ostheimer[54] believe that a great aid to the physician is the instruction of mothers by nurses in the home, in the care, clothing and diet of infants. Levy[55] shows how poor milk and unscientific feeding keep the infant mortality high. With better milk and more scientific feeding, infant mortality should decrease. Freeman[56] describes the good work accomplished by the Infant Milk Depots in New York City.

FATS

Chapin[57] states that, while both fats and carbohydrates furnish energy and supply water to the infant metabolism, the fats supply more of the water than do the carbohydrates. This water results from the combination of the hydrogen from the fat with the oxygen from the blood. In milk feeding, the fats supply the greater part of the metabolic water. Brady[16] notes that infants show great differences in their tolerance to fats and carbohydrates. Stolte[58] makes a plea for the use of higher fats instead of such high sugars. He has given from 5 to 7 per cent. fat, he claims, with good results. When fatty soaps are found in the stools, he adds milk sugar. Americans, however, will prefer the views of Eaton,[59] who reports eight infants gaining steadily in growth and weight on low fats, with comparatively high proteids. He admits that there are individual children who will do better on higher fats with lower proteins. It is most important to feed each infant as an individual. Morse[14] advises relatively high fat and sugar with relatively low protein. Glaze-

brook[60] never feeds high fat mixtures. Towle and Talbot,[61] who studied fat and carbohydrate indigestion in infants with acute inflammatory eczema, conclude that as all babies with indigestion do not develop eczema, some underlying systemic condition must exist in those in whom eczema appears. Huldschinsky[62] shows that the amount of volatile acids remaining in the stomach contents of overfed infants with gastro-intestinal disease is in direct proportion to the fat content of the food. These volatile acids do not injure a healthy stomach. They can only harm a stomach whose functions have already been affected.

CARBOHYDRATES

Brady[63] has written an interesting report of 170 institution babies fed on a mixture containing barley, dextrin, maltose and cane-sugar, with a total carbohydrate per cent. of 7.91. While ninety babies were breast-fed for two weeks, forty-five took this polycarbohydrate mixture from birth. Eighteen infants died, a mortality of 10½ per cent. Brady[16] notes elsewhere that carbohydrates have the greatest influence in advancing the weight curve. Stolte[53] says that, although high sugar may cause gain in weight, the weight rapidly decreases if the baby develops even a mild infection. Johnson[64] reports diarrhea in three bottle-fed infants of 4, 7 and 9 months, in whom an excess of milk-sugar was the evident cause. He advises using mixtures without sugar at first, and later adding dextrimaltose. Torian[47] believes that sugar should compose 6 or 7 per cent. of the food. He advises no starch except a cereal diluent until the age of 1 year; then he begins starch gradually. Calvary[65] considers from 3 to 5 per cent. of sugar generally enough in an infant food. For well infants he prefers cane-sugar. For those with a tendency to diarrhea or during convalescence from gastro-enteritis, he uses dextrin and maltose; for those with a tendency to constipation, milk-sugar. While the baby cannot gain without enough sugar, too much sugar may cause severe intestinal disturbance. Southworth[66] considers combined dextrin and maltose of value in disturbances of digestion. Both milk and cane-sugar are still to be used, however. Morse[67] considers lactose preferable to maltose under ordinary conditions, as it is more slowly broken down and has, therefore, a more prolonged influence. The dextrin-maltose preparations can often be given earlier than lactose in diarrhea; but when the *Bacillus perfringens* and the gas bacillus cause acid stools, maltose is more harmful than lactose. Calvary[68] states that milk sugar is of as much value as the other sugars as regards gain in weight. Coit[29] lays emphasis on the use of a clean milk sugar, prepared from whey from fresh high grade milk. Crookshank[8] advises cereals when the baby reaches 7 months. Pfersdorff and Stolte[69] show how well starches are assimilated in gruel form. Chapin[70] prefers to use cereal gruels by

converting the starch into dextrin or maltose by means of diastase. St. Philippe[8] believes that constipation, with slowly developing gastro-intestinal dyspepsia, is the principal danger from the abuse of cereals in infancy. Chapin[57] shows that the organism obtains the water from carbohydrates as a product of the splitting up of the carbohydrate. Salge[71] observed a rapid loss of salts occur when an infant is fed on cereal foods. The composition of the blood changes, which is an unfavorable prognostic sign. The disturbances in the retention of salts and water by the organism explains the slight resistance of such children to infections. Meyer[72] noted that babies fed on concentrated milk without water lose weight, but gain again when water is given. Samelson[73] tried to demonstrate that the so-called "salt fever" was due to bacterial toxins. Strauss[74] considers the "salt fever" due more to the sodium than to the chlorin *ion*. Von Reuss[7] insists that infants need water soon after birth. The so-called "inanition fever" generally disappears when water is given freely.

PROTEIN

Holt and Levene[75, 76] noted fever just as long as high protein diet was continued. They believe that 4.5 per cent. protein should never be given, except with whey. This confirms Vaughan's statement of fever following the subcutaneous injection of protein. Brown[77] and Wollstein[78] discuss changes noted in infants on high proteins. Eaton[59] noticed that infants did very well on low fats and comparatively high proteins. Torian[47] says that about 10 per cent. of the total caloric value of the feeding should be protein. Stolte[58] says that no baby will gain on high protein without enough sugar in addition.

ALBUMIN MILK: BUTTERMILK

Brady[62] reports that twelve among 170 asylum infants who did not gain on the polycarbohydrate mixture, did well on albumin milk. He considers a high protein necessary, though the fat is only to be increased when the baby fails to gain in weight. Friedlander and Greenbaum[79] state that sugar will be well tolerated after a period on albumin milk. Johnson[64] uses albumin milk in subacute and chronic diarrhea. Weston[80] considers albumin milk valuable in reducing infant mortality, especially in the fermental disorders of infancy. Brady[15] states that it is of value, like Keller's malt-soup, because of its low fat, high protein and maltose. Cassel and Weill and Mouriquond are not enthusiastic about albumin milk. Cassel[81] noted that it was not well taken by all infants and that the gain in weight was not steady. Weill and Mouriquond[82] tried it on sixteen infants, ten of whom were ill. None died, while all the others lost weight and strength, had vomiting and digestive disturbances, and diarrhea developed in the healthy babies. Neff[83] gave a high percentage

albumin milk, made with buttermilk, with good results. In diarrhea, Morgan[84] prefers albumin milk in infants under 3 months; in others he uses buttermilk, after all acute symptoms have disappeared. Stolte[22] and Schaps[85] report good results in diarrhea by feeding babies on buttermilk. Stolte[86] reports four cases in which he gave buttermilk in place of two or more regular feedings each day with success. He ascribes the good results to the low fat and the high lime and high protein content of the buttermilk. It is of value in infants who have lost much weight when they do not gain on breast milk alone. Peiser[87] describes an alkalinized kefir with which he claims success in acute intestinal disturbances in infants.

DESICCATED MILK

The controversy on the value of dry milk powder in infant feeding is still agitating French physicians. Aviragnet, Bloch-Michel and Dorlencourt, champions of this form of infant food, report twenty infants who did well. They consider desiccated milk better than any other form of milk for infants. Nageotte-Wilbouchewitch[89] agrees with their conclusions. But Variot, Zuber, Lavialle and Sédillot,[90] who observed fourteen infants, found that only six gained in weight. Diarrhea developed commonly. Variot[91] insists that it is the cause of grave gastro-intestinal disturbance. Variot, Lavialle and Rousselot[92] also report using sweetened condensed milk in twenty babies who vomited, with cessation of the vomiting. It is not to be given to atrophic infants; nor do they advise it when cow's milk can be obtained. Ausset[93] considers condensed milk of little value.

FEEDING AFTER FIRST YEAR

Yerington[94] advises four daily feedings after the baby reaches 1 year. He allows milk, well-cooked cereals and vegetables, zweiback or toast, scraped beef, baked potato, baked apple and apple sauce. Later he adds meats, finely cut up, soft-boiled or poached egg, custard, gelatin, etc. Many infants are overfed on fats and starches. Ostheimer[5] prefers three daily meals; with meat, beef juice and broths only for weak, undernourished infants. He lays emphasis on teaching every baby to masticate its food. Küsel[95] also states that all food requires thorough mastication. Nourishing food, containing calcium and magnesium salts, is needed early, such as cheese, milk, eggs, green vegetables and the natural mineral waters. Starch is worse than sugar as the exciting cause of dental caries. An excess of sour fruit or acids also has a decalcifying action on the teeth. Hutchison[96] lays especial emphasis on defective mastication and excessive carbohydrates as causes of dyspepsia. Dennett[97] advises three bottles and two meals for infants from 12 to 15 months of age. He thinks that babies of this age are fed too often, and are, therefore, never hungry. They frequently get too much cereal, soup and sugar.

Watson[98] ascertained by chemical analysis the food requirements of healthy children about 5 years old. He advises not to attach too much importance to the chemical composition of the food. In gouty children protein should be restricted; in tuberculous children, it should be increased. Food should be pure and hard enough to necessitate thorough mastication. Sugars and sweets should be restricted. Langstein and Kassowitz[99] recommend green vegetables and fresh fruits, even as early as seven months of age. They also advise powdered spinach and carrots, prepared with flour, sugar and milk.

FEEDING PREMATURE AND ATROPHIC INFANTS

Durham[100] considers breast milk essential for premature infants; also external heat and pure air. He reports a case, born at twenty-six weeks, weighing 1 pound, 12 ounces at birth. At 8 months she died of pneumonia. Rott[101] uses a No. 9 or No. 10 catheter, 6 inches long, introduced into the esophagus when the infant is too weak to nurse. He feeds the baby from five to ten times in twenty-four hours. Of eighteen infants fed in this way only two died. He keeps them in an incubator until they weigh 2,000 grams. Wyatt[102] reports eight successful cases of feeding premature infants. He begins on the third day with equal parts of cow's milk and a mixture containing water, cream, lactose and sodium citrate. He gives an ounce every two hours and more as soon as the baby will take it. Litzenberg[103] advocates four-hour intervals for premature infants. The relatively large amounts of nourishment required can be given without trouble. He reports fifteen cases with increase in weight, more sleep and less digestive disturbances by this method. Dennett[104] found that emaciated, atrophic infants required 150 to 170 calories per kilogram, while normal babies did well on 110 to 120 calories per kilo. Gray[105] keeps these infants warm, wrapped in flannel soaked with cod liver oil.

Little[106] considers a wet-nurse the best treatment for marantic infants, whose condition is often the direct result of improper feeding. Dunn[107] studied the feeding of infants with malnutrition, for four years. He first gives a fat-free food, with maltose and high casein, adding fat if the stools improve, but the baby fails to gain in weight. He prefers maltose to lactose. Glazebrook[60] believes that thymus gland may help atrophic infants. Morse[108] has reported four cases of what he calls intestinal toxemia in the new-born, a bacterial infection of the meconium, occurring twenty-four to forty-eight hours after birth, with the absorption of toxic products. On castor oil, withdrawal of all food and the administration of water, recovery follows. He also irrigates the bowels at the beginning. Breast milk should then be given. If cow's milk is given, it should contain much lactose, to change bacterial activity from the proteolytic to the fermentative type.

DISEASES OF THE DIGESTIVE SYSTEM

ESOPHAGUS

Morse[109] reports three cases of congenitally imperforate esophagus. He also describes two cases of stricture, with dilatation, and two cases of cardiospasm. Brennemann[110] reports three cases of congenital atresia. Shukowsky and Baron[111] report a case, the esophagus ending in a blind sac, from which there existed a fistula into the trachea. Haus[112] reports performing an emergency operation, median esophagostomy, in a baby of 14 months, who had swallowed a garter-clasp, 1½ inches long. Theories to explain the origin of stenosis of the esophagus and intestine are discussed by Forssner[113, 114] and Kreuter.[115]

MUCOUS GASTRITIS

Cautley[116] discusses the treatment of mucous gastritis in infancy; diet and alkalies. When much mucus is secreted, lavage with an alkaline solution is advised. As food he uses sweet whey powder, 1 dram in 2 ounces of water; later he adds cream; then substitutes peptonized milk, and then milk and barley water. He prefers milk-sugar in these cases. In the habitual vomiting of infants, Sébilleau[117] advises a tablespoonful of a 1.7 per cent. sodium citrate solution before each nursing or in each bottle.

CYCLIC VOMITING

Sandt[118] considers cyclic vomiting a neurosis, found only in nervous children, often closely related to migraine. It is primarily due to the effect of certain exhausting or depressing influences on the nervous mechanism of digestion; the intestinal toxemia is secondary, possible only through the subsequent perverted function of the liver. In treatment attention should be directed to the general physical well being of the child rather than to the alimentary tract. Langmead[119] considers cyclic vomiting due to some disturbance in metabolism. The cause may be a toxemia or infection derived from stomach or intestines, since gastro-intestinal lesions are found *post mortem;* constipation or diarrhea is a precursor; marked indican reaction is found in the urine and decided changes in the liver. The possible danger of an anesthetic to children with cyclic vomiting should never be forgotten. Terrien[120] suggests that some adrenal condition may be the cause. In a child under 4 years of age, autopsy showed the liver large and fatty. To a child of 3 years he gave 1/1,000 of epinephrin by enema and vomiting ceased. When he gave this to a boy of 8 years twice each month, it apparently prevented recurrence.

GASTRIC ULCER

Cackovic[121] states that 2.32 per cent. out of 172 cases of gastric ulcer operated on at Zagreb, occurred in children under 10 years of age and 7.55 per cent. in children under 15. These figures are probably too low,

since gastric ulcer must have existed early in many others, though diagnosis was only made later. In thirteen cases, with symptoms which appeared early in life, operation was performed years later; all except two recovered. Comby[122] reports five cases of rumination in children between 2 and 4 years. To overcome this habit he advises toning up the nervous system and regulating digestion. Hutchison[96] advises change of air, corrected diet and hygiene in the treatment of the dyspepsias of childhood.

PYLORIC OBSTRUCTION

Cowie[123] has studied the significance of the "pyloric reflex." As the opening of the pylorus may be delayed by giving a normal infant an alkali, giving more alkali to an infant with hyperchlorhydria will effect the same result. The dose needed can be regulated by analyzing the stomach contents. Rapid recovery followed the controlled use of the alkali in Cowie's case, a baby of 8 months. The protein in the baby's food was cautiously increased, reaching nearly 2 per cent. three months after beginning treatment. Theoretically, high protein would be advisable in pyloric spasm or stenosis with hyperchlorhydria. Recovery means that conditions have been produced which are favorable to normal pyloric function. Pisek and Le Wald[124] exhibit twenty-four radiographs showing in cases of pylorospasm that food had already passed into the duodenum a few minutes after ingestion. When this is not found, true pyloric stenosis exists and the case needs surgical treatment. They advise radiographic study of every case of pyloric obstruction. Taylor[125] reports three attacks of pylorospasm in a boy of 2 years; Fischer[126] a case in a child of 14 weeks, with recovery on large and small doses of breast milk. Schäfer[127] reports a fatal case of pyloric stenosis, with the development of "hour-glass" stomach. Molloy[128] reports a similar condition in a baby 4 days old. Rammstedt[129] reports three cases of pyloric stenosis. He prefers Weber's pyloroplasty to gastro-enterostomy. Keefe,[130] who reports operating on babies of 4 and 5 weeks, also considers pyloroplasty the operation of choice. Lendon[131] did posterior gastro-enterostomy in a boy of 6 weeks. Fredet and Tixier[132] report gastro-enterostomy on a baby of 13 days, with recovery. Hipsley[133] examined six cases *post mortem*. While gastric dilatation was absent in four, three of whom had died after operation, it was found in the other two which had not come to operation. Louria[134] reports an interesting case of sudden projectile vomiting of a large amount, with great abdominal pain, rapid and severe distention, collapse, constipation and visible peristalsis due to acute dilatation of the stomach complicating pneumonia in a boy of 2 years. The patient recovered.

DUODENAL ULCER

Flesch[135] diagnosed duodenal ulcer during life in a baby of 3 months from the profound anemia and bloody stools. He believes that the lack of salts and inanition due to improper food led to autodigestion of the mucous membrane of the duodenum. He quotes Entz, who found eleven cases of duodenal ulcer in infants between 6 weeks and 6 months among 364 autopsies at the Children's Hospital, Budapest. Studies of the pancreatic ferments have been made by Hess[136, 137, 138] by means of the duodenal catheter. The duodenal catheter has also been used in the diagnosis and treatment of pylorospasm by Putzig[139] and Hess.[136] The value of radiography in the diagnosis of duodenal ulcer is commented on by Pisek and Le Wald,[124] Means[140] and Cole.[141]

INTESTINES

Rosenstern[142] observed that infants commonly fail to take enough food. He therefore recommends more frequent feeding. To some infants a stronger food may be given, with water by rectal or subcutaneous injection if there is diarrhea. Cautley[143] and Hutchison[96] discuss the treatment of intestinal dyspepsia. Abt[144] reviews the classification of gastro-intestinal diseases in ancient and modern times. Johnson[145] explains the development of the mucous membrane of the large intestine and vermiform process in the human embryo.

CONSTIPATION

That constipation is a common cause of gastro-intestinal disturbance in childhood is shown by Freeburg.[146] Grulee[147] advises against the routine use of cathartics in these cases, but advises suppositories. Massage may be of benefit. Weill[148] insists on regular daily bowel movements before the child goes to school. Bueri[149] advises using hot fomentations which regulate the circulation, combat congestion and stimulate function in the intestines.

DIARRHEA

Brady[16] states that summer diarrhea is now explained as due to overstepping the tolerance limit, which has been lowered by excessive heat. By careful observation of the weight curve, temperature and general behavior of the infant, we can determine the tolerance limit. The causes of summer diarrhea in Spain are given by Suñer[150] as overfeeding, artificial feeding, food spoiled by heat, digestion lowered by heat, diminished bactericidal strength of intestinal secretions, increased bacterial growth due to heat and transmission of pathogenic germs by flies. As prophylaxis he recommends decreasing quantity of food during hot weather, encouraging breast feeding, aseptic artificial feeding, avoiding exposure to heat, and the use of screens. Morton[151] considers the heat and the food the causes of summer diarrhea. Heim[152] blames it on spoiled milk and salted

broths. The baby must be protected from the heat, given plenty of water and no salt soups. Royster[130] has collected statistics to show a definite relation between the mortality from summer diarrhea and the humidity rather than the heat alone. MacCarthy[154] and Venable[155] consider the food the chief cause of summer diarrhea. Allan[156] lays emphasis on flies, Bloch[157] reports a low mortality, 100 infants dying among 3,000 sick. Though fifty-eight were very ill, only six died. He gives water only, as long as alarming symptoms exist. He begins feeding with much diluted milk and gradually increases its strength. Rulison[158] begins with diluted skimmed milk. While all writers advise stopping food at once, Filiatreau[159] has had success on that alone. Initial doses of castor oil are advised by Morton[151] and Heiman.[160] Johnson[64] says that drugs are not usually needed, though Connelly[161] uses them. Morse[8] uses alkalies in gastric, but not in intestinal indigestion. The treatment of ileocolitis is discussed by Botts;[162] of cholera infantum by Hobbs.[163] Whey is advised by Frank;[164] albumin milk by Morgan,[84] Heiman[151] and Abt;[165] buttermilk by Schaps,[85] Stolte[86] and Abt;[165] alkalinized kefir by Peiser.[87] Clock[166] reports wonderful success in twenty-two cases of bottle-fed infants from 5 weeks to 10 months of age, seen in private practice, to whom tablets of the *Bacillus lactis bulgaricus* were given before and after feedings, while the food was not stopped in one case. Macé and Quinton[167] use the subcutaneous injection of sterile sea water with marvellous results. Schmidt[168] reports success with the insufflation of oxygen into the rectum and stomach in six out of nine infants so treated. Abt[165] discusses the abuse of cathartics, starvation and loss of heat. Long continued laxatives produce gastro-enteritis; even heart weakness may result from active purgation. Babies are often kept on too low a diet too long, and therefore show diminished resistance to infection. Sick babies must be kept warm. Overfeeding during the period of reconstruction and repair is as pernicious as starvation.

JAUNDICE

From a study of the "hematies granuleuses" of Cesar Demel in twenty-eight new-born infants, Maliwa[169] concludes that when these colored erythrocytes are found in large numbers, even though the baby is not jaundiced, icterus will probably develop; but when only a few are present, no jaundice will appear. In the former case jaundice develops as the erythrocytes disappear rapidly. Niemann[170] reports tests made on an infant with congenital stenosis of the bile-ducts, who died at nine months. Serious symptoms only appeared during the last month. The bile in infants has the same significance in fat absorption as in adults. Koplik and Crohn,[171] in a case of congenital absence of the bile ducts, found nitrogen metabolism normal, but poor absorption of fat and defi-

cient splitting up of the neutral fats. Blakeway[172] reports a most interesting case of congenital absence of the gall-bladder, associated with imperfect pancreatic development and imperforate anus. Niemann[173] considers catarrhal jaundice an infection, most common in Autumn. Huber[174] reports a case of acute yellow atrophy in a child of three years, ill only two weeks. Peiper[175] reports a case of malignant adenoma in a girl of 8 months, an overgrowth of embryonic tissue. Idzumi[176] reports a case of primary cancer of the liver in a boy of 7 months.

APPENDICITIS

A large number of articles have recently appeared on appendicits in children. Stiven[177] considers operation imperative in children. If not operated on within twenty-four hours, infection becomes general. Complications are less frequent in children than in adults. Deaver[178] reports operating on 100 children. Fifty-six had been vigorously purged; of these, 86 per cent. showed serious lesions; 12 had diffuse peritonitis. There was perforation in 22, gangrene in 15, acute ulceration in 6, periappendicular abscess in 4, acute catarrhal inflammation in 8, with an indefinite condition in 1. Among the 44 children who had not been purged before admission, only one case of perforation occurred and 8 cases of gangrene. Only one died. Cumston[179] says the prognosis is poor, because the condition is not recognized or is only recognized too late. Sonnenburg[180] considers chronic appendicitis more frequent than acute. The common symptoms are constipation, headache and digestive disturbances. Colitis is often due to appendicitis. He advises prompt removal of the appendix. Lereboullet[181] also discusses chronic appendicitis. Fowler[182] reports 179 cases of acute appendicitis in children under 12, with operation. Twenty died. Cautley[183] cautions the physician about calling in a surgeon at once in every doubtful case. Mitchell[184] operated on 40 children between 14 months and 13 years of age. Ten died. Clarke,[185] among 33 cases, had 9 children under 12, two of whom died. Mummery[186] has never seen acute appendicitis in children under 5 years. Wade[187] believes more children are affected than is commonly estimated. Attacks are either very mild or very severe. Both Mummery and Wade advise early operation. Wahle[188] reports what he considers appendicitis in a girl of 3 and a boy of 5 years, within one week. He believes this was an acute infection. Both recovered without operation. Fairbanks[189] says that appendicitis is much more apt to lead to abscess or general peritonitis in children than in adults. Thread-worms play a not unimportant part in the etiology. Green[190] reports three cases in which seat-worms were found in the appendix at operation. Rheindorf[191] concludes that oxyuris is found in a large percentage of cases of appendicitis in children. Brun[192] found ascarides in the appendix in three cases at herniotomy.

He advises postponing all operations if a preliminary purgative discloses the presence of ascarides. Remsen[193] operated on an infant 16 days old for appendicitis occurring in the sac of an inguinal hernia. Becker[194] found appendicitis in an incarcerated left-sided inguinal hernia in an infant of two months. He says this is much more rare than on the right side. Neuwalt[195] reports operating on a girl of 8 months for intussusception with appendectomy. Five months later laparotomy was performed again for adhesions. The baby recovered after each operation.

INTESTINAL OBSTRUCTION

Edmunds[196] considers congenital malformations the common cause of acute intestinal obstruction in children, especially absence of the greater part of the intestine, imperforate anus and Meckel's diverticulum. Other causes may be tuberculosis, cysts, hernia, foreign bodies and appendicitis. Children stand laparotomy poorly. Mummery[186] discusses acute obstruction, appendicitis and intussusception. Early diagnosis and early operation are advised. Kock and Oerum[197] report 397 cases of intussusception in children, 288 in infants under a year. They also advise early operation. Woolfenden[198] reports performing enterostomy for intussusception in a boy of 3 months twenty-seven hours after the onset of symptoms, with recovery. Horsley[199] reports three cases in children under 1 year and one in a child of 3 years. Barling[200] reports operating for intussusception in a boy of 4 years, who had Henoch's purpura, with recovery. Power[201] showed a boy of 5½ years, with irreducible intussusception due to sarcoma. He recovered after the operation, but a week later a mass was found near the right nipple. Gaardlund[202] reports two cases of invagination, one in a boy of 14 years, with Meckel's diverticulum, recovering after operation; the other in a girl of 4 months, ending fatally. Three cases of intussusception with Meckel's diverticulum are reported by Brennecke.[203] Grigsby[204] reports three cases of imperforate anus with one death. Weber[205] operated on a baby 15 days old with atresia of the duodenum, with death seven hours later. Bell and Le Wald[206] report a case of chronic colitis in a child of 3 years, with great deformity of the sigmoid, as shown by radiographs. Aitken[207] reports an unique case of doubling of the intestine, occurring in an infant with a large congenital umbilical hernia, extensively ulcerated, who died when 2 days old. The ileum bifurcated, forming two separate tubes of equal caliber, 17 cm. long, ending on opposite sides of a conical cecum, which had a deep longitudinal sulcus and a small appendix 2 cm. long at the end of each side. Smester[208] reports thirty years of success in the treatment of umbilical hernia in infants by piling up strips of adhesive plaster after reducing the hernia and approximating the edges of the ring. Over this plaster he paints collodion. Elting[209] reports a case of fistula in ano in a baby

of 10 months. He has seen a second case in a child under 10 years. Vidakovich[210] describes an adhesive plaster bandage over the buttocks for preventing prolapse of the rectum. Von Sury[211] tells of a baby dying twenty-four hours after having an enema given by a midwife when but 12 hours old. The autopsy showed the intestine ruptured, with peritonitis. The author believes that rupture occurred before birth, yet many readers will consider the enema the cause.

INGUINAL HERNIA

Ochsner[212] believes that approximately 95 per cent. of inguinal hernias in childhood will heal spontaneously if the intra-abdominal pressure is relieved and the sac kept empty. Rest in bed is better than a truss. Operation is indicated in strangulated hernia, irreducible hernia due to adhesions, congenital hernia if unusually large, irreducible hydrocele and in some cases of undescended testicle. Meredith[213] and Savariaud[214] advise early operation. Nicholls[215] reports operating on a strangulated hernia in an infant of 8 weeks with recovery. Collins[216] reports a similar case. Appendicitis in a hernia sac is described by Remsen[193] and by Becker.[194]

INTESTINAL FLORA

After thorough investigation, Veeder, Kilduffe and Denny[217] found dysentery micro-organisms in about 20 per cent. of cases of ileocolitis in infants. Dysentery organisms are only present as a predominating type in severe cases, and it is in these cases that the distinctive lesions of dysentery are found *post mortem*. Friedlander and Greenbaum[79] found the same intestinal flora in infants when fed on ordinary milk mixtures and on albumin milk. The latter, given for a short period, causes acidophilic to replace putrefactive organisms and rests the gastro-intestinal tract by reason of its low sugar content. Simonds[218] reports an epidemic of thirty-four cases of bacillary dysentery. Twelve died, all under 4 years, eight before the sixth day. Graham[219] reports a case of bacillary dysentery, Type Y, in a 6-months old infant in India, with recovery after three injections of Shiga serum. Meilo Leitao[220] found 7 cases of flagellate dysentery among 32 children under 10 years of age. The youngest was 2 months old, breast-fed. It is a benign affection, not dependent on the food. Murphy[221] found intestinal worms in only seven out of 102 children between 2 and 12 years of age, whose stools he examined. Eosinophilia was not constant. Rémy[222] describes the Triboulet and Pettenkofer methods of examining feces, with a discussion of the results obtained by each method.

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Jour. Missouri Med. Assn., January, 1913.

AMERICAN PEDIATRIC SOCIETY

The annual meeting of the American Pediatric Society was held in Washington, D. C., May 5 to 7, 1913. The following officers were elected for the coming year:

President, Samuel McC. Hamill, M.D., Philadelphia.
Vice-President, Matthias Nicoll, Jr., M.D., New York.
Secretary, Samuel S. Adams, M.D., Washington, D. C.
Treasurer, Charles Hunter Dunn, M.D., Boston.
Recorder and Editor, L. E. LaFétra, M.D., New York.
Assistant Editor, O. M. Schloss, M.D., New York.
Member of Council, D. M. Cowie, M.D., Ann Arbor.

The following were elected to membership in the Society:

Julius Parker Sedgwick, M.D., Minneapolis.
Herbert B. Wilcox, M.D., New York.
B. Raymond Hoobler, M.D., New York.
Henry John Gerstenberger, M.D., Cleveland.
Alfred Friedlander, M.D., Cincinnati.

ENGLISH CONFERENCE ON INFANT MORTALITY

An English-speaking Conference on the Prevention of Infant Mortality will be held in Caxton Hall, Westminster, London, on Monday morning, Monday afternoon and Tuesday morning, Aug. 4 and 5, 1913. The meetings will be held under the auspices of the (British) National Association for the Prevention of Infant Mortality and The Welfare of Infancy under the patronage of the King and Queen, and will convene immediately preceding the opening of the International Medical Congress.

A tentative program has been issued by the committee which indicates that the papers will consist largely of medical opinion. The subjects treated will be:

The responsibility of central and local authorities in infant and child hygiene.

The administrative control of the milk-supply.

The necessity for special education in infant hygiene.

Medical problems in infant nutrition.

Antenatal hygiene.

The president of the conference will be the Hon. John Burns, M. P., president for the Local Government Board. The chairman of the English Executive Committee is Sir Thomas Barlow and the secretary, Miss J. Halford, 4 Tavistock Square, London, W. C.

The American committee, in charge of the part to be taken by the United States and Canada, will furnish information to those desiring to attend the conference.

Dr. Henry L. Coit, chairman, 277 Mt. Prospect Avenue, Newark, N. J.

Dr. Philip Van Ingen, secretary, 125 East Seventy-First Street, New York City.

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